LONGEVITY IN THE 21ST CENTURY

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ABSTRACT

The main objective of this paper is to offer a detailed analysis of mortality change in the United Kingdom at the beginning the 21st century. Starting from an exploration of 20th century mortality trends, focusing in particular on the 1990s, underlying forces driving trends in longevity are discussed. These include the 'cohort effect' and the 'ageing of mortality improvement.' International mortality statistics and trends are also analysed. The pace of medical advances is discussed with specific focus on research into the ageing process and a potential treatment for cardiovascular disease. The paper also discusses the potential threat from infectious diseases.

The analysis of underlying trends suggests that life expectancy in retirement in the U.K. is likely to increase rapidly in the early part of the 21st century. Some scientists are also claiming that we will be seeing the fruits of anti-ageing research within just a few decades.

A core theme of the paper is that future projections should be grounded in as good an understanding of the past as possible. Different methods for projecting future rates of mortality are discussed and it is noted that emphasis should be placed on the uncertainty surrounding projections.

The financial impact of using different assumptions for future mortality is explored. Significant differences in the cost of an annuity or pension arise from the use of the various projection bases.

Life assurance companies have already declared significant losses as a result of strengthening reserves on annuity portfolios. Taken together, future increases in life expectancy, increasing awareness of the risk of providing longevity insurance, changes in legislation and shortages in market capacity and capital, may well lead to worsening annuity rates.

It is difficult to assess the precise impact of future changes in life expectancy on final salary pension schemes. There is a lack of readily available information on the mortality assumptions being used in practice. It is therefore suggested that more disclosure in this area would be helpful. Employers sponsoring final salary schemes are making promises to their employees that extend up to 70 or 80 years into the future. Actuaries should be clear in spelling out to employers and trustees the nature of the risks behind the promises they are making. Future scheme design should reflect the possibility of substantial increases in life expectancy.

An over-riding implication of the anticipated increases in life expectancy is that people will remain in work for longer in the future. The age at which people retire will inevitably have to increase and this trend will necessarily drive changes in all aspects of our society. As actuaries we have a vital role in helping to inform the wider debate.

KEYWORDS

Mortality; Longevity; Ageing; Annuities; Cohort Effect

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1. Introduction

1.1 *Introduction*

1.1.1 As a subject for research, human longevity is vast topic. Mortality rates are bare statistics that reflect the evolution of our society. During the 20th century huge increases in life expectancy have followed medical and scientific breakthroughs mostly unimagined a hundred years ago.

1.1.2 Some scientists are now predicting enormous extensions in our potential lifespan and mortality tables published in recent years have included substantial allowances for future improvement. Mortality improvements in the 21st century could have enormous social and financial implications.

1.1.3 The purpose of this paper is to analyse how mortality in the United Kingdom is likely to change in future years. Underpinning the report is a desire to draw out the key trends and the major forces that are likely to drive future shifts in mortality.

1.1.4 Whilst the analysis of past experience covers all adult ages, the later sections of this paper focus more on the mortality of older people and in particular life expectancy in retirement. This particular emphasis has been chosen because the implications of mortality change at higher ages are currently of particular interest to life assurance companies and pension schemes.

1.1.5 A cross board working party was established to produce this paper, which has been sponsored by the Social Policy Board. The working party was formed, partly to provide a review of mortality matters for the actuarial profession, and partly to help to counter criticisms from outside the profession that actuaries have been failing to keep up with likely changes in life expectancy.

1.1.6 This paper takes forward some of the ideas originally outlined in 'Mortality in the Next Millennium' (Willets, 1999) and reflects the latest mortality data and recent developments in medicine, demography and actuarial science. However, the intention is to substantially build upon the work of the earlier paper, rather than simply provide an update.

1.2 *Contents*

1.2.1 The paper has been divided into six main sections:

— *Section 2, 20th Century Trends*: a discussion of how and why mortality rates in the U.K. have changed over the course of the 20th century, and in particular during the 1990s. Five key forces behind the recent changes are identified.

— *Section 3, International Experience*: an analysis of how U.K. mortality experience and improvement rates compare with data from other countries.
— *Section 4, Medical Advances*: a discussion of how advances in medical science are likely to impact the pace of future improvements in longevity. The section focuses on two main areas: potential developments in the treatment of heart disease and research into the ageing process.
— *Section 5, The Threat from Infectious Diseases*: an examination of possible threats to the health of our society: including AIDS, new variant CJD, antibacterial resistance and emerging diseases.
— *Section 6, Projecting the Future*: a discussion of the likely pattern of future mortality improvement and different methods of mortality projection. This section makes reference to a working paper by the Continuous Mortality Investigation (C.M.I.) Bureau Working Party on mortality projection due to be published early in 2004. In addition to this, various projections are compared, including the interim C.M.I. cohort bases published in 2002 and the Government Actuary's Department (GAD) basis issued in December 2003.
— *Section 7, Implications*: an assessment of the likely implications of future mortality change for life assurance companies, general insurers, pension schemes, society in general and the actuarial profession.

1.3 *Appendices*

The paper also has two appendices which contain more detailed information on two particular topics. Appendix 1 explores recent trends in mortality in England and Wales by cause of death. Appendix 2 examines the threat from infectious diseases in detail.

2. 20th Century Trends

2.1 *Introduction*

2.1.1 The purpose of this section is to describe how mortality in the U.K. has changed during the 20th century. The analysis concentrates mainly on death rates for adults, since the mortality of children has a fairly limited impact on pensions and life insurance. In many instances, population mortality data for England and Wales, rather than for the whole of the U.K., has been used. This reflects the fact that figures are often more readily, or only, available in this format.

2.1.2 Past trends are analysed, with particular emphasis placed on describing the changes that have occurred during the 1990s and outlining the major factors influencing mortality change at the beginning of the 21st century. Five key forces behind the recent improvements are identified.

2.2 *A Brief Overview of the 20th Century*

2.2.1 Over the course of the 20th century mortality in the U.K. has improved to an amazing degree. We now live an entirely different kind of life and think of death in a completely different way to those alive at the beginning of the previous century.

2.2.2 In 1901 life expectancy at birth in England and Wales was 46 years for males and 50 years for females (O.N.S., 1997). By 2001 — just a century on — these figures had increased to 76 and 81 years respectively (GAD, 2003).

2.2.3 At the beginning of the 20th century 16% of new-born boys and 13% of new-born girls would not survive their first year of life. By 2001 both these figures had reduced to well under 1% (GAD, 2003). Moreover, in 1901 only 36% of males and 43% of females could expect to reach age 65. By 2001 these survival rates had increased to 83% and 89% respectively (GAD, 2003).

2.2.4 We are now unfortunate not to reach retirement age. In fact most of us probably *expect* to reach our 60s, 70s or even 80s. Table 2.2a illustrates the extent to which mortality rates improved between 1901 and 2001.

2.2.5 The improvements have clearly been substantial at all ages, although the youngest ages have seen the greatest reductions. The 98% reduction for 5-year-olds means that the mortality rate for this age group is now just 2% of the rate at the beginning of the 20th century. In fact, there is a very clear correlation between age and the degree to which mortality has improved. For elderly people, especially men, the improvement has been more modest, though still significant.

2.2.6 The most important factor driving these mortality improvements was the conquest of infectious diseases. This was the single biggest health success of the 20th century. At the turn of the century diseases such as tuberculosis, typhoid, measles, scarlet fever and diphtheria exacted a terrible toll. This is illustrated by Table 2.2b, which shows the proportion of all deaths, at various ages, due to infectious diseases.

Table 2.2a. Reduction in mortality rate between 1901 and 2001, England and Wales population

	Reduction in mortality rate	
Age	Male	Female
5	98%	98%
25	82%	92%
45	80%	83%
65	63%	71%
85	37%	49%

Own figures — data source: ELT7 (1901-10) and GAD Interim Life Tables 2000-02

Table 2.2b. Proportion of all deaths due to infectious diseases in the period 1901 to 1910 and for 2001, England and Wales population

	Proportion of deaths due to infectious diseases			
	1901-10		2001	
Age group	Male	Female	Male	Female
1-14	43%	47%	6%	6%
15-44	46%	49%	2%	3%
44-64	16%	11%	<1%	<1%
65 and over	4%	5%	<1%	<1%

Own figures — data source: O.N.S. (1997a) and O.N.S. (2003)

2.2.7 The development of vaccines and the introduction of antibiotics after the Second World War has almost eliminated deaths from this cause. Infectious diseases now account for fewer than 1.0% of all deaths (O.N.S., 2003).

2.2.8 The figures in Table 2.2b show why the reduction in deaths from infectious diseases has had far more effect on aggregate mortality rates for younger ages. Infectious diseases simply accounted for a much greater share of total deaths for younger people. This partly explains the age pattern seen in Table 2.2a.

2.2.9 Tables 2.2c and 2.2d show how life expectancy, calculated on the basis of life tables reflecting mortality for each of the periods shown, with no allowance for future improvements, has increased over the 20th century. Over the first half of the century, mortality improvements were strongest for children and young adults, and generally higher for females than males. Life expectancy at birth increased by 17.9 years for males and 19.1 years for

Table 2.2c. Improvements in life expectancy over the 20th century, England and Wales population, males

	Total life expectancy on reaching the ages shown				
Year	at birth	at age 15	at age 45	at age 65	at age 80
1901-10	48.5	62.3	68.3	75.8	84.9
1910-12	51.5	63.6	68.9	76.0	84.9
1920-22	55.6	65.1	70.2	76.4	84.9
1930-32	58.7	66.2	70.5	76.3	84.7
1940-42					
1950-52	66.4	69.4	71.5	76.7	84.9
1960-62	68.1	70.3	72.1	77.0	85.2
1970-72	69.0	70.8	72.4	77.2	85.5
1980-82	71.0	72.3	73.7	78.0	85.8
1990-92	73.4	74.3	75.7	79.3	86.4
2000-02	75.9	76.6	78.0	81.0	87.1

Own figures — data sources: O.N.S. (1997a), G.A.D. (2003a) and English Life Tables

Table 2.2d. Improvements in life expectancy over the 20th century England and Wales population — females

Year	Total life expectancy on reaching the ages shown				
	at birth	at age 15	at age 45	at age 65	at age 80
1901-10	52.4	65.1	70.5	77.0	85.4
1910-12	55.4	66.4	71.3	77.4	85.5
1920-22	59.6	68.1	72.7	77.9	85.6
1930-32	62.9	69.3	73.3	78.1	85.5
1940-42					
1950-52	71.5	74.0	75.8	79.3	85.8
1960-62	74.0	75.9	77.1	80.3	86.4
1970-72	75.3	76.8	77.9	81.1	87.0
1980-82	77.0	78.0	79.0	82.0	87.5
1990-92	79.0	79.7	80.5	83.1	88.4
2000-02	80.6	81.1	81.9	84.1	88.7

Own figures — data sources: O.N.S. (1997a), GAD (2003a) and English Life Tables

females; life expectancy on reaching age 15 increased by 7.1 years for males and 8.9 years for females. In contrast, life expectancy on reaching age 65 only increased by 0.9 years for men and 2.3 years for women, reflecting only small improvements in the mortality of elderly people.

2.2.10 Over the second half of the century, mortality improvements have shifted along the age range. In this period, life expectancy on reaching age 65 increased by 4.3 years for men and 4.8 years for women, reflecting much greater rates of mortality improvements for the elderly. In addition, increases in life expectancy have been roughly similar for males and females.

2.2.11 The last 30 years, in particular, show a very different pattern to that of the first half of the 20th century. Mortality improvements since 1970 have been strongest in the over 45s and life expectancy for men has been increasing faster than that of women. Life expectancy at birth increased by 6.9 years for males and 5.3 years for females; life expectancy on reaching age 45 increased by 5.6 years for males and 4.0 years for females. The rate of increase in life expectancy on reaching ages 65 and 80 has been increasing rapidly for men.

2.3 *The Last Four Decades*

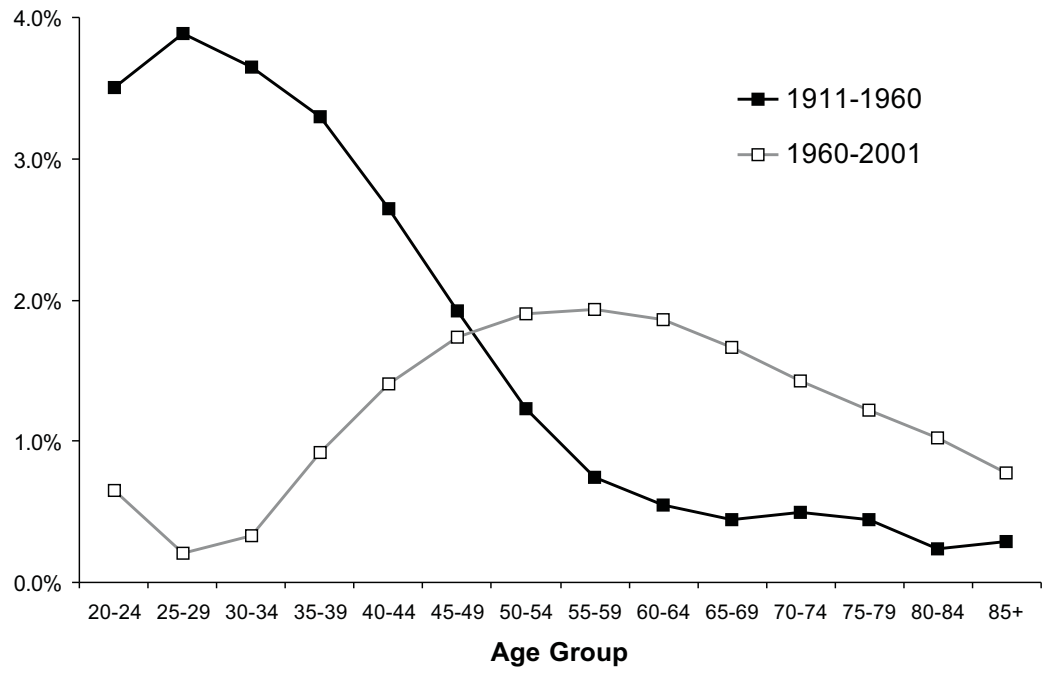
2.3.1 Given this change over time in the pattern of mortality improvement, some sections of the paper focus on trends emerging over the last 40 years or so. Figures 2.3a and 2.3b give another illustration of how the age groups showing the greatest 'mortality improvements' have shifted upwards. Throughout this paper the term 'mortality improvement' is used to signify the reduction in the rate of mortality for a given age from one year to the next. The mortality rates used are central, rather than initial, rates. So,

the mortality improvement rate for age x in calendar year $t = 1 - m_{x,t}/m_{x,t-1}$. Where multi-year periods are reported, average annual improvement rates have been calculated using log-linear regression, unless otherwise stated. In the period 1911 to 1960 rates of mortality improvement were very high for people in their 20s, 30s and 40s, but tailed away with increased age. Between 1960 and 2001 rates of improvement fell (from the 1911-60 levels) for younger adults, but were higher for older people.

2.3.2 As our society grows increasingly mature, and life expectancy increases, the phenomenon of mortality improvement also seems to be 'ageing.' The ages showing the greatest improvements are steadily moving upwards. This idea is explored in some depth in Section 2.18.

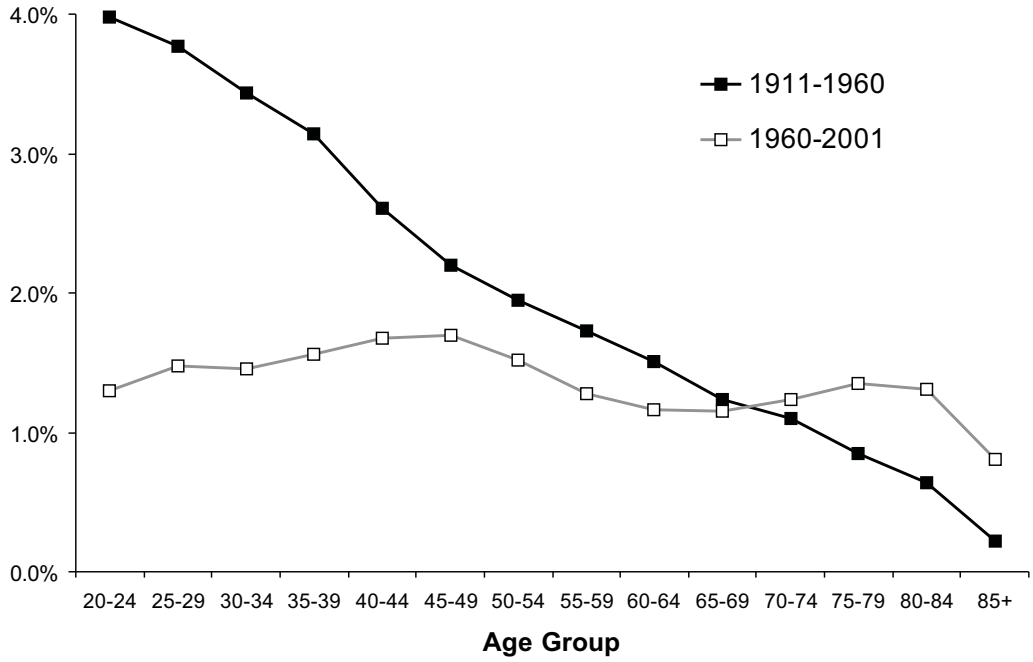
2.3.3 Over the period 1960 to 2001 average annual mortality improvement rates (given in Table 2.3) have exceeded 1.0% p.a. for women of almost all ages and for men between the ages of 40 and 84. In most cases, average improvement rates have been between 1.0% p.a. and 1.9% p.a. A simple average for all ages and both sexes combined is 1.3% p.a.

2.3.4 Before exploring the changes in mortality seen during the 1990s, some background into the various causes of death is given in Sections 2.4 to 2.6.



Own figures — data source; O.N.S. (2003)

Figure 2.3a. Average annual mortality improvement rates, England and Wales population, 1911-2001, males



Own figures — data source; O.N.S. (2003)

Figure 2.3b. Average annual mortality improvement rates, England and Wales population, 1911-2001, females

Table 2.3. Average rate of annual mortality improvement, England and Wales population, 1960 to 2001

	Average rate of annual mortality improvement	
Age group	Male	Female
20-24	0.7%	1.3%
25-29	0.2%	1.5%
30-34	0.3%	1.5%
35-39	0.9%	1.6%
40-44	1.4%	1.7%
45-49	1.7%	1.7%
50-54	1.9%	1.5%
55-59	1.9%	1.3%
60-64	1.9%	1.2%
65-69	1.7%	1.2%
70-74	1.4%	1.2%
75-79	1.2%	1.3%
80-84	1.0%	1.3%
85 and over	0.8%	0.8%

Own figures — data source: O.N.S. (2003)

2.4 *How Mortality Rates Vary by Age*

2.4.1 The primary causes of death vary enormously by age. In order to appreciate the different forces affecting mortality improvement rates, it is useful to see how the constituents of mortality rates change with age.

Table 2.4. Central mortality rates by age and sex, England and Wales population, 2001

Age group	Number of deaths per 10,000 population Male	Female
20-24	8	3
40-44	19	12
60-64	128	79
80-84	954	656

Own figures — data source: O.N.S. (2003)

2.4.2 However, before we look at cause of death, it is helpful to remember the extent to which *aggregate* mortality rates vary by age. Table 2.4 contains some example central rates of mortality for the population of England and Wales.

2.4.3 The degree to which mortality rates increase with age is evident. A woman aged in her early 80s is about 220 times more likely to die in the next year than one in her early 20s.

2.4.4 Moving to cause of death, Tables 2.5 and 2.6 show how mortality varied by cause in 2001. This information is readily available from the Office of National Statistics (O.N.S., 2003) as cause of death is recorded on death certificates. Each cause of death is assigned a code from the International Classification of Diseases (I.C.D.). The C.M.I. Bureau investigation into cause of death has been closed — the last experience published was for the period 1991-94. As a consequence, in these sections (as in many others) we have solely considered population data.

2.4.5 There are a number of well-documented issues with the use of cause of death data. The subjective element in the assignment of a cause of death and changes in practice and classification can influence trends over time. Having said that, these difficulties (if suitably allowed for) do not invalidate inferences which can be drawn from cause of death statistics.

Table 2.5a. Deaths by cause, England and Wales population, 2001, males, deaths per 100,000 lives

Cause of death	Age group 20-29	30-39	40-49	50-59	60-69	70-79	80+
Infectious diseases	2	3	5	5	10	30	91
Cancers	7	17	59	227	655	1,531	2,817
Circulatory	5	17	71	215	668	2,028	5,685
Respiratory	2	5	10	35	138	574	2,256
Other health related	19	29	55	88	167	492	2,047
Violence and accidents	48	48	42	37	34	63	207
All causes	83	119	241	606	1,671	4,719	13,104

Own figures — data source: O.N.S. (2003)

Table 2.5b. Deaths by cause, England and Wales population, 2001, males, percentage of deaths attributable to each cause

	Age group						
Cause of death	20-29	30-39	40-49	50-59	60-69	70-79	80+
Infectious (excl AIDS)	2%	2%	1%	1%	1%	1%	1%
AIDS	0%	1%	1%	0%	0%	0%	0%
All infectious	2%	3%	2%	1%	1%	1%	1%
Prostate cancer	0%	0%	0%	1%	3%	4%	5%
Lung cancer	0%	1%	4%	9%	11%	9%	4%
Other cancers	8%	13%	20%	27%	25%	20%	13%
All cancers	8%	14%	24%	37%	39%	32%	22%
Heart disease	1%	6%	19%	25%	27%	26%	22%
Stroke	2%	2%	4%	5%	6%	9%	12%
Other circulatory	3%	5%	6%	6%	7%	8%	10%
All circulatory	6%	14%	30%	35%	40%	43%	43%
Chronic lung disease	0%	0%	1%	2%	4%	6%	5%
Pneumonia	1%	2%	2%	2%	2%	3%	9%
Asthma	0%	0%	1%	0%	0%	0%	0%
Other respiratory	1%	2%	1%	2%	2%	3%	3%
All respiratory	2%	4%	4%	6%	8%	12%	17%
Dementia	0%	0%	0%	0%	0%	1%	3%
Diabetes	0%	1%	1%	1%	1%	1%	1%
Drugs or alcohol	11%	6%	2%	1%	0%	0%	0%
Epilepsy	2%	2%	1%	0%	0%	0%	0%
Liver disease	1%	6%	10%	6%	2%	1%	0%
MS	0%	0%	0%	0%	0%	0%	0%
'Old age'	0%	0%	0%	0%	0%	0%	2%
Other health related	9%	9%	8%	6%	6%	7%	9%
All other health related	23%	24%	22%	14%	9%	10%	15%
Motor vehicle accidents	18%	9%	3%	1%	0%	0%	0%
Other accidents	13%	10%	5%	2%	1%	1%	1%
Suicide	15%	13%	6%	2%	1%	0%	0%
Violence	12%	8%	3%	1%	0%	0%	0%
All violence and accidents	58%	41%	17%	6%	2%	1%	2%

Own figures — data source: O.N.S. (2003)

2.5 *Cause of Death for Men*

2.5.1 All-cause mortality rates rise by around 5% compound for each year of age for men in their 30s, increasing to 11% for each year of age for men aged 60+.

2.5.2 The majority of deaths for younger males are non-health-related — that is, violent or accidental. Specifically, for men in the 20-29 age group

58% of deaths are violent or accidental, and only 11% are due to the 'big three' killers: cancer; heart disease; and stroke. The main causes of death for this age group are motor vehicle accidents (18%) and suicides (15%), followed by other accidents (13%), violence (12%) and drugs or alcohol (11%).

2.5.3 The absolute death rate from violence and accidents falls slightly with increasing age for men between their 20s and their 60s. So, with increasing age, the percentage of violent or accidental deaths falls away rapidly — the figure dropping to just 2% for men in their 60s. Conversely, the proportion of deaths due to health-related causes, notably cancer and heart disease, increases. For men between their 20s and their 60s, deaths from circulatory disorders increase by around 13% compound for each year of age and deaths from cancer by 12%.

2.5.4 Cancer reaches its relative peak (as a proportion of deaths) in the 50 to 69 age range, causing 38% of all deaths; the most prevalent form being lung cancer.

2.5.5 Circulatory disorders (which include heart disease and strokes) cause approximately 40% to 45% of deaths for people aged 60 and over.

2.5.6 The mortality rates from these causes, and particularly from cancers, increase less quickly with age for men aged 60 and over. However, deaths from respiratory disorders rise very rapidly with age (15% compound for each year of age after 60), and so the cause pattern for men in their 80s and above is markedly different to men in their 60s. Relatively speaking, cancer and heart disease are less significant, with strokes and respiratory disorders (especially pneumonia) more important.

2.6 *Cause of Death for Women*

2.6.1 All cause mortality rates for females rise by around 8% compound for each year of age up to the 40s, increasing to 10% for each year of age through to the 60s and to 14% for each year of age for women in their 80s.

Table 2.6a. Deaths by cause, England and Wales population, 2001, females, deaths per 100,000 lives

	Age group						
Cause of death	20-29	30-39	40-49	50-59	60-69	70-79	80+
Infectious diseases	1	2	2	4	8	25	75
Cancers	7	23	74	214	473	965	1,587
Circulatory	3	8	26	75	299	1,210	4,936
Respiratory	1	2	7	24	97	370	1,664
Other health related	10	15	33	60	128	412	2,535
Violence and accidents	10	11	14	15	16	41	197
All causes	32	62	156	392	1,021	3,023	10,993

Own figures — data source: O.N.S. (2003)

Table 2.6b. Deaths by cause, England and Wales population, 2001, females, percentage of deaths attributable to each cause

	Age group						
Cause of death	20-29	30-39	40-49	50-59	60-69	70-79	80+
Infectious (excl AIDS)	3%	2%	1%	1%	1%	1%	1%
AIDS	1%	1%	0%	0%	0%	0%	0%
All infectious	4%	3%	1%	1%	1%	1%	1%
Breast cancer	2%	14%	16%	15%	8%	4%	2%
Cervical cancer	2%	4%	3%	1%	1%	0%	0%
Lung cancer	0%	1%	5%	9%	9%	7%	2%
Other cancers	17%	18%	23%	30%	28%	21%	10%
All cancers	20%	38%	47%	54%	46%	32%	14%
Heart disease	0%	3%	6%	9%	16%	20%	18%
Stroke	3%	5%	6%	5%	7%	11%	16%
Other circulatory	5%	6%	5%	5%	7%	9%	11%
All circulatory	8%	13%	17%	19%	29%	40%	45%
Chronic lung disease	0%	0%	1%	2%	5%	6%	3%
Pneumonia	2%	2%	2%	2%	2%	4%	10%
Asthma	1%	1%	1%	1%	0%	0%	0%
Other respiratory	2%	1%	1%	2%	2%	2%	2%
All respiratory	5%	3%	5%	6%	9%	12%	15%
Dementia	0%	0%	0%	0%	0%	2%	5%
Diabetes	1%	1%	1%	1%	1%	1%	1%
Drugs or alcohol	4%	2%	1%	0%	0%	0%	0%
Epilepsy	4%	2%	1%	0%	0%	0%	0%
Liver disease	1%	5%	8%	4%	2%	1%	0%
MS	0%	1%	2%	1%	0%	0%	0%
'Old age'	0%	0%	0%	0%	0%	0%	6%
Other health related	20%	13%	9%	8%	8%	10%	11%
All other health related	30%	24%	22%	14%	11%	14%	23%
Motor vehicle accidents	8%	3%	1%	0%	0%	0%	0%
Other accidents	7%	4%	3%	1%	1%	1%	2%
Suicide	8%	5%	2%	1%	0%	0%	0%
Violence	9%	6%	2%	1%	0%	0%	0%
All violence and accidents	32%	18%	9%	4%	2%	1%	2%

Own figures — data source: O.N.S. (2003)

2.6.2 The deaths by cause analysis for females also shows a significant proportion of deaths due to violence or accidents at young ages, again falling away rapidly with age. However, both the proportion of all deaths and the absolute death rate accounted for by violence and accidents are very much lower than are the case for males.

2.6.3 Also, as was the case for males, the causes of death leading to the steepest increase in mortality rates with age for women under age 50 are cancer and circulatory disease. For women in their 30s death rates from cancer increase by around 14% compound for each year of age.

2.6.4 Cancer dominates as a proportion of deaths in the 40 to 69 age range, causing around 50% of all deaths, and peaking at a younger age than for men. Breast cancer is the biggest killer, accounting for 16% of all deaths in the 40-49 age group.

2.6.5 Circulatory disorders grow as a proportion of deaths with increasing age, but are relatively less important than for males, not reaching 40% until age 70+. As for men, heart attacks account for more deaths than strokes, although the difference is far smaller for women.

2.6.6 For women over age 60, mortality rates from cancers increase less and less quickly with rising age. However, deaths from circulatory and respiratory disorders continue to rise very rapidly at 15% compound for each year of age. Accordingly, the cause pattern for women in their 70s and above is markedly different than that for younger women. Whilst still increasing in absolute terms, cancer deaths fall away rapidly as a proportion of all deaths. The dominant causes of death for women aged 80+ are heart attack, stroke, pneumonia, and other circulatory and respiratory disorders.

2.6.7 An alternative way of looking at the sex differentials is to further consider the absolute rates of mortality by cause of death, as shown in Tables 2.5a and 2.6a. Table 2.6c shows the ratio of absolute mortality rates by sex, for each age band and major cause group.

2.6.8 The largest relative mortality differentials between the sexes are for deaths due to violence and accidents and to circulatory diseases. Mortality rates are closer for cancer but are notably higher for females than for males in the 30 to 49 age range. The pattern changes somewhat at the oldest ages with the percentage differential reducing as mortality rates from circulatory

Table 2.6c. Mortality rates for females as a proportion of mortality rates for males, England and Wales population, 2001, by age group and by cause of death

	Age group						
Cause of death	20-29	30-39	40-49	50-59	60-69	70-79	80+
Infectious diseases	87%	57%	42%	79%	87%	82%	82%
Cancers	94%	137%	126%	94%	72%	63%	56%
Circulatory	52%	48%	37%	35%	45%	60%	87%
Respiratory	79%	44%	70%	69%	70%	64%	74%
Other health related	50%	53%	61%	69%	77%	84%	124%
Violence and accidents	22%	24%	33%	41%	48%	64%	95%
All causes	39%	52%	65%	65%	61%	64%	84%

Own figures — data source: O.N.S. (2003)

Table 2.6d. England and Wales population, 2001, deaths per 1,000 population

Age group	Violence and accidents			Health-related		
	Male	Female	Ratio of female to male	Male	Female	Ratio of female to male
20-29	0.48	0.10	22%	0.35	0.22	63%
30-39	0.48	0.11	24%	0.71	0.51	72%

Own figures — data source: O.N.S. (2003)

Table 2.6e. England and Wales population, 2001, deaths per 1,000 population

Age group	Cancer			Heart disease		
	Male	Female	Ratio of female to male	Male	Female	Ratio of female to male
40-49	0.59	0.74	126%	0.47	0.09	19%
50-59	2.27	2.14	94%	1.51	0.35	23%

Own figures — data source: O.N.S. (2003)

and respiratory diseases, and accidents, rise sharply for females, relative to males, and more than outweigh the relative fall in cancer mortality rates for females.

2.6.9 Two particular features are worthy of further comment. Table 2.6d shows that, at younger ages, men are over four times more likely than women to die from violent or accidental causes. The sex differential for health-related deaths is much narrower, but still significant.

2.6.10 For females, cause of death is very heavily weighted towards cancer. Heart disease is a noticeably less-common form of death for middle-aged women compared with middle-aged men. The true extent of this can again be appreciated by looking at the absolute figures.

2.6.11 It can be seen from Table 2.6e that male and female cancer mortality rates for ages 40-59 are similar, but male heart disease mortality rates are four to five times greater than those for females. This factor alone goes a long way to explaining the sex differentials in mortality rates at this age.

2.7 *Mortality Improvement in the 1990s*

2.7.1 Section 2.3 briefly described how rates of mortality improvement for older adults have increased since the 1960s. The pace of the most recent improvements can be illustrated by considering how quickly mortality has improved during the 1990s. More specifically, changes over the period from

Table 2.7a. Average annual rates of mortality improvement, England and Wales population, 1989-2001

	Age group						
	20-29	30-39	40-49	50-59	60-69	70-79	80+
Male	−0.1%	−0.1%	0.8%	2.7%	3.4%	2.2%	0.9%
Female	0.4%	0.7%	0.8%	2.1%	2.8%	1.4%	0.0%

Own figures — data source: O.N.S. (2003)

1989 to 2001 are considered. Table 2.7a gives annualised rates of improvement up to 2001.

2.7.2 There has been little overall change in mortality rates for men in their 20s and 30s, reasonably modest improvements for women of these ages and for both men and women in their 40s, but very substantial improvements for the 50-79 age group, peaking at ages 60-69. In fact, the mortality of people in their 60s has never improved so rapidly.

2.7.3 For the oldest adults, mortality has not declined quite so quickly (although the 2.2% p.a. rate for men aged 70-79 is also an historical high). Improvements for the '80 plus' age group have been dampened down, to some extent, by the fact that the average age of those above 80 has been increasing.

2.7.4 A closer inspection of the most recently published mortality rates at older ages actually shows an accelerating improvement trend. Tables 2.7b and 2.7c illustrate this by showing some improvement rates (fitted using log linear regression) for successive five-year periods. The rate of improvement for males and females has clearly increased in recent years.

Table 2.7b. Average annual rate of mortality improvement over successive five-year periods, males in the population of England and Wales aged 60-89

Age group	1990-1994	1994-1998	1998-2002
60-69	3.0%	3.2%	3.9%
70-79	1.8%	2.3%	3.7%
80-89	1.1%	1.2%	2.5%

Own figures — data source: GAD

Table 2.7c. Annualised rates of mortality improvement over successive five-year periods, females in the population of England and Wales aged 60-89

Age group	1990-1994	1994-1998	1998-2002
60-69	2.4%	2.5%	3.1%
70-79	1.0%	1.0%	3.0%
80-89	0.8%	0.4%	1.7%

Own figures — data source: GAD

2.8 *Mortality Improvement by Cause*

2.8.1 The tables presented in this section of the paper show a breakdown by cause of death of the mortality improvement rates over the period 1989 to 2001 shown in Table 2.7a. In other words, the changes in aggregate, all-cause mortality produced by changes in death rates for individual causes are shown. For instance, Table 2.8a shows that mortality rates for men in their 50s improved by 2.71% p.a. in total. Furthermore, improvements in heart

Table 2.8a. Breakdown of contributions to overall average annual mortality improvements over the period 1989-2001, by cause, England and Wales population, males

	Age group						
Cause of death	20-29	30-39	40-49	50-59	60-69	70-79	80+
Infectious (excl AIDS)	−0.03%	0.01%	−0.03%	−0.01%	0.00%	−0.01%	−0.02%
AIDS	0.12%	0.33%	0.12%	0.02%	0.00%	0.00%	0.00%
All infectious	0.10%	0.34%	0.08%	0.01%	0.00%	−0.01%	−0.02%
Prostate cancer	0.00%	0.00%	0.00%	0.02%	0.05%	0.07%	−0.01%
Lung cancer	0.00%	0.08%	0.24%	0.41%	0.57%	0.29%	0.15%
Other cancers	0.16%	0.26%	0.31%	0.34%	0.31%	0.10%	0.06%
All cancers	0.16%	0.34%	0.55%	0.77%	0.92%	0.45%	0.20%
Heart disease	0.02%	0.42%	1.05%	1.86%	1.81%	1.20%	0.63%
Stroke	−0.01%	0.07%	0.05%	0.16%	0.27%	0.36%	0.30%
Other circulatory	−0.03%	−0.12%	−0.10%	0.04%	0.10%	0.04%	0.00%
All circulatory	−0.02%	0.36%	1.01%	2.05%	2.18%	1.61%	0.93%
Chronic lung disease	0.00%	0.00%	−0.01%	0.01%	0.16%	0.02%	0.08%
Pneumonia	−0.03%	0.06%	−0.11%	−0.10%	−0.22%	−0.07%	−0.43%
Asthma	0.05%	0.05%	0.01%	0.02%	0.03%	0.00%	0.00%
Other respiratory	−0.01%	−0.01%	0.00%	0.03%	0.16%	0.03%	0.07%
All respiratory	0.02%	0.09%	−0.11%	−0.04%	0.13%	−0.01%	−0.27%
Dementia	0.00%	0.00%	0.00%	0.01%	0.01%	0.04%	0.05%
Diabetes	−0.01%	−0.02%	−0.02%	0.04%	0.03%	0.04%	0.05%
Drugs or alcohol	−0.68%	−0.49%	−0.15%	−0.03%	0.00%	0.00%	0.00%
Epilepsy	0.02%	−0.04%	−0.02%	−0.01%	0.00%	0.00%	0.00%
Liver disease	−0.04%	−0.28%	−0.50%	−0.20%	−0.05%	−0.01%	0.00%
MS	0.00%	0.01%	0.01%	0.00%	0.00%	0.00%	0.00%
'Old age'	0.00%	0.00%	0.00%	0.00%	0.00%	−0.01%	−0.21%
Other health related	−0.08%	−0.14%	−0.12%	0.02%	0.09%	0.11%	0.18%
All other health related	−0.78%	−0.96%	−0.80%	−0.18%	0.09%	0.17%	0.07%
Motor vehicle accidents	0.66%	0.05%	0.09%	0.04%	0.02%	0.02%	0.01%
Other accidents	−0.20%	−0.17%	−0.04%	0.01%	0.01%	−0.01%	0.02%
Suicide	0.21%	−0.02%	0.06%	0.04%	0.01%	0.01%	0.00%
Violence	−0.24%	−0.12%	−0.03%	0.01%	0.01%	0.00%	0.00%
All violence and accidents	0.43%	−0.25%	0.09%	0.10%	0.04%	0.02%	−0.01%
All causes	−0.10%	−0.08%	0.81%	2.71%	3.35%	2.22%	0.89%

Own figures — data source: O.N.S. (2003)

Table 2.8b. Breakdown of contributions to overall average annual mortality improvements over the period 1989-2001, by cause, England and Wales population, females

	Age group						
Cause of death	20-29	30-39	40-49	50-59	60-69	70-79	80+
Infectious (excl AIDS)	−0.09%	−0.05%	−0.03%	−0.01%	−0.01%	−0.02%	−0.02%
AIDS	0.00%	−0.06%	−0.01%	0.00%	0.00%	0.00%	0.00%
All infectious	−0.09%	−0.11%	−0.04%	−0.01%	−0.01%	−0.02%	−0.02%
Breast cancer	0.03%	0.27%	0.64%	0.49%	0.27%	0.09%	0.03%
Cervical cancer	0.07%	0.43%	0.17%	0.09%	0.05%	0.02%	0.00%
Lung cancer	0.00%	0.04%	0.03%	0.06%	0.27%	−0.09%	−0.03%
Other cancers	0.20%	0.36%	0.41%	0.50%	0.30%	0.03%	0.03%
All cancers	0.30%	1.10%	1.25%	1.14%	0.90%	0.05%	0.04%
Heart disease	0.00%	0.14%	0.15%	0.78%	1.29%	1.13%	0.58%
Stroke	0.03%	0.22%	0.03%	0.17%	0.35%	0.49%	0.30%
Other circulatory	−0.09%	−0.33%	−0.11%	0.02%	0.06%	−0.01%	0.03%
All circulatory	−0.06%	0.03%	0.08%	0.97%	1.70%	1.61%	0.92%
Chronic lung disease	0.00%	0.00%	−0.01%	0.01%	0.00%	−0.22%	−0.09%
Pneumonia	−0.04%	−0.14%	−0.14%	−0.08%	−0.05%	−0.32%	−0.45%
Asthma	0.04%	0.04%	0.03%	0.03%	0.01%	0.02%	0.00%
Other respiratory	−0.01%	−0.03%	−0.01%	0.02%	0.03%	0.01%	−0.04%
All respiratory	−0.01%	−0.13%	−0.13%	−0.03%	−0.01%	−0.50%	−0.58%
Dementia	0.00%	0.00%	0.00%	0.01%	0.02%	0.05%	0.02%
Diabetes	0.02%	−0.01%	0.01%	0.07%	0.05%	0.08%	0.04%
Drugs or alcohol	−0.29%	−0.12%	−0.06%	−0.02%	0.00%	0.00%	0.00%
Epilepsy	0.05%	0.00%	−0.03%	0.00%	0.00%	0.00%	0.00%
Liver disease	−0.09%	−0.17%	−0.31%	−0.15%	−0.02%	0.00%	0.00%
MS	0.03%	0.08%	0.00%	−0.01%	0.01%	0.00%	0.00%
'Old age'	0.00%	0.00%	0.00%	0.00%	0.00%	−0.02%	−0.48%
Other health related	−0.01%	−0.12%	−0.07%	0.07%	0.12%	0.12%	0.10%
All other health related	−0.28%	−0.36%	−0.45%	−0.02%	0.18%	0.22%	−0.32%
Motor vehicle accidents	0.50%	0.15%	0.07%	0.04%	0.02%	0.02%	0.01%
Other accidents	0.00%	−0.08%	−0.04%	−0.02%	0.01%	0.00%	−0.03%
Suicide	−0.05%	0.03%	0.05%	0.01%	0.02%	0.01%	0.00%
Violence	0.07%	0.05%	0.02%	0.03%	0.01%	0.01%	0.00%
All violence and accidents	0.52%	0.16%	0.10%	0.07%	0.06%	0.03%	−0.02%
All causes	0.38%	0.69%	0.80%	2.11%	2.80%	1.38%	0.02%

Own figures — data source: O.N.S. (2003)

disease mortality were responsible for two-thirds of this change. The heart disease improvements *alone* would have lead to 1.86% p.a. reductions in all-cause mortality for this age group. It should be noted that there is an element of approximation in the methodology used to derive the contributions by cause. Two decimal places are shown only to give some differentiation between the many smaller contributions.

2.8.2 A detailed analysis of mortality change over the period from 1989 to 2001 is given in Appendix 1, where graphs showing trends in specific causes of death are given, and reasons for the trends are discussed. A summary of some of the major trends is given in Sections 2.9 to 2.12.

2.9 *Mortality Improvements for Adults in their 20s and 30s*

2.9.1 There has been little overall change in mortality during the 1990s for people in their 20s and 30s. However, the relative stability in aggregate rates belies substantial shifts in deaths from individual causes.

2.9.2 There were very significant improvements in mortality from motor vehicle accidents. Reduced deaths from this cause alone would have produced 0.7% p.a. improvements in all-cause mortality for men, and 0.5% p.a. for women, in the 20 to 29 age group.

2.9.3 There were also substantial improvements, for both men and women, in mortality from cancer, heart disease and stroke, especially for those in their 30s. For men in their 30s there was also a material contribution from a reduction in AIDS deaths.

2.9.4 However, these improvements were offset by increased deaths for some causes, most notably from the abuse of drugs and alcohol and from liver disease. Other causes for mortality deterioration for young adults are violence and (non-vehicle) accidents (men), infectious disease and AIDS (women), and some circulatory and respiratory conditions (both sexes). Increased deaths related to drugs, alcohol and liver disease contribute 0.7% p.a. deterioration to all-cause mortality for men in these age groups and 0.4% p.a. for women. Deaths from these causes have been increasing at a frighteningly rapid pace: during the 1990s, deaths from drugs and alcohol quadrupled for men aged 20-39 and more than doubled for women of the same age group. Further detail is given in Appendix 1.

2.9.5 Overall, the negative trends slightly outweighed the positive for men, resulting in slight deteriorations in experience. The opposite was the case for women, where the particularly strong contribution from reduced cancer deaths shifted the overall balance to one of modest improvements.

2.10 *Mortality Improvements for Adults in their 40s*

2.10.1 For men and women in their 40s, there were still significant opposing forces in mortality change. However, the causes producing improvements have outweighed those resulting in increased deaths, giving modest overall reductions in mortality.

2.10.2 Improvements came mainly from cancer and heart disease. Deteriorations arose principally from liver disease and the abuse of drugs and alcohol. Although the average all-case mortality improvements were similar for men and women in this age group, the balance of the underlying drivers was quite different.

2.10.3 For men, the biggest contribution came from reduced mortality

from heart disease. Reduced deaths from this cause alone would have produced 1.05% p.a. improvements in all-cause mortality for men aged 40-49. The effect was much weaker for women in this age group.

2.10.4 For women, improvements in cancer mortality dominate contributing 1.25% p.a. to all-cause mortality improvements for this age group. A major factor was increased awareness of, and screening for, breast and cervical cancer.

2.11 *Mortality Improvements for Adults in their 50s and 60s*

2.11.1 The most dramatic mortality improvements in the 1990s applied to men and women in their 50s and 60s. There were very significant reductions in deaths from both circulatory disease and cancer. Also, for these age groups and in contrast to the younger ages, there were few material causes showing increases in mortality rates.

2.11.2 The figures shown in Appendix 1 clearly show that mortality from heart disease, cancer and strokes — that is, the 'big three' killers — has fallen very steadily. There is very little sign that this trend is slowing down. Mortality from heart disease has reduced particularly quickly. Over the 12-year period from 1989 to 2001 the reduction at some ages has been as much as 50%.

2.11.3 For men in their 50s and 60s, reductions in heart disease mortality *alone* would have led to 1.8% p.a. improvements in aggregate mortality. Factors behind these improvements include reductions in cigarette smoking, improvements in diet, medical advances and increasing prescription of (more effective) drugs for cardio-vascular disease. The next most important cause leading to mortality improvements for males has been cancer. Reduced deaths from this cause have also stemmed from less smoking, along with improved treatments and diagnostic techniques.

2.11.4 For females, the improvements in heart disease mortality have been less significant, relative to males, although still important. This is largely because heart disease is a far less significant cause of death for females (see Table 2.6e). The dominant source of mortality improvements for women in their 50s and 60s was reduced deaths from cancer, reflecting improved treatments and the benefit of the screening programme introduced for breast cancer in the early 1990s.

2.12 *Mortality Improvements for Adults in their 70s and Above*

The improvements in cancer and circulatory disorder mortality have had proportionately less of an impact on all-cause rates at age 70 and above, with the effect progressively reducing with advancing age. This is certainly not because these causes are uncommon at these ages. For instance, for people in their 70s, cancer and circulatory disorders together account for 75% and 72% of all deaths for men and women respectively; even for those in their 80s and beyond, these proportions only reduce to 65% and 59%

respectively. Instead, rates of mortality from these causes have simply not improved as quickly over the 1990s for people in their 70s and 80s as they have done for people aged in their 50s and 60s. The figures in Appendix 1 go some way towards illustrating this point.

2.13 *Saving Lives*

2.13.1 In July 1999 the government published the White Paper *Saving Lives: Our Healthier Nation* (Department of Health, 1999). The paper outlined various ways in which the health of the U.K. population could be improved.

2.13.2 Four priority areas were established, and specific targets were defined for each, namely:

— a reduction in the death rate from heart disease, stroke and related illnesses amongst people under age 75 by at least 40% by the year 2010;
— a reduction in the death rate from accidents by at least 20% by the year 2010;
— a reduction in the death rate from cancer amongst people under age 75 by at least 20% by the year 2010; and
— a reduction in the death rate from suicide and undetermined injury by at least 20% by the year 2010.

2.13.3 The implication is that the Government clearly hopes that there will be further substantial improvements in mortality from major causes of death. If the targets *were* met, and there were no changes in mortality from other causes of death, then the resulting mortality improvement rates would be as follows (assuming the target improvements were exactly met at all ages):

Table 2.13. Minimum rates of annual mortality improvement targeted by the White Paper *Saving Lives*, 1997 to 2010, England and Wales population

	Age group					
	20-29	30-39	40-49	50-59	60-69	70-79
Male	1.1%	1.3%	1.7%	2.1%	2.2%	2.1%
Female	1.0%	1.3%	1.5%	1.7%	1.9%	2.0%

Own figures — data sources: O.N.S. (2003), Department of Health (1999)

2.13.4 Significant progress has already been made, particularly in reducing deaths from circulatory diseases and from cancer (Department of Health, 2003).

— From a base of 141.5 in 1995-97 for the relevant population segment, death rates per 100,000 from circulatory disease have fallen to 108.5 for 2000-02, a 23% fall in just the first five years of a 14-year target period.

— For cancer, the equivalent figures are 141.4 per 100,000 in 1995-97 falling by 10% to 126.8 for 2000-02. As for circulatory disease, death rates have moved just over half way towards target in well under half the working timeframe.

2.14 *Five Key Forces*

2.14.1 Up to this point, the paper has largely concentrated on describing past changes in mortality experience, especially during the 1990s. Rates of mortality improvement have been given and the causes behind the improvements described. Some of the risk factors driving the changes have also been discussed.

2.14.2 The remaining sections of this part of the paper will concentrate on bringing out the 'big themes' — the key forces driving mortality change at the beginning of the 21st century. Five areas will be discussed in more detail, namely:

— the 'cohort effect';
— the 'ageing of mortality improvement';
— increased uncertainty at younger ages;
— changes in the prevalence of cigarette smoking; and
— widening social class differentials.

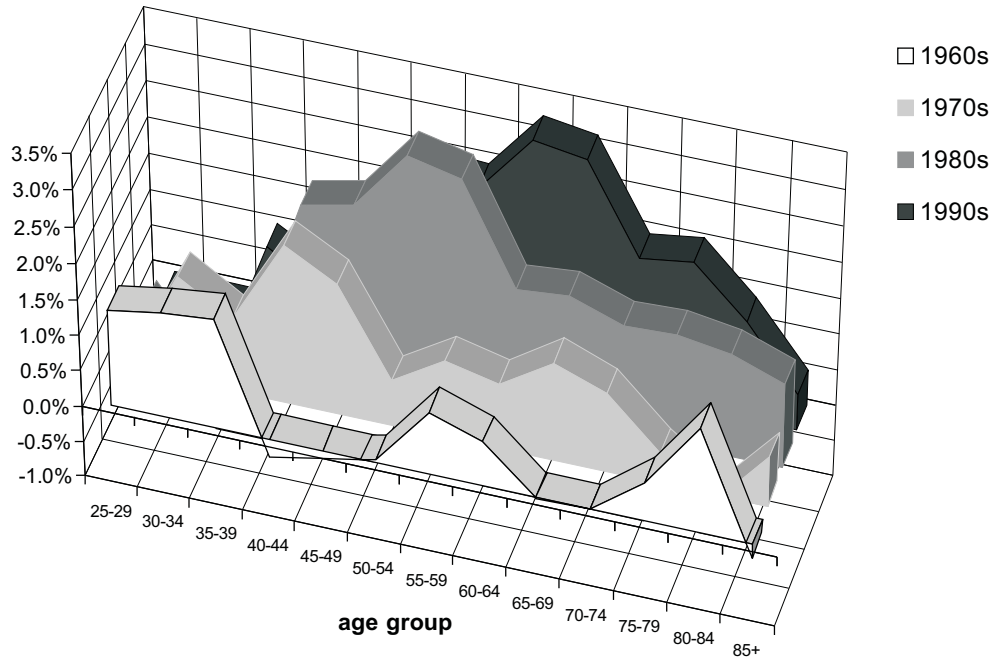
2.15 *The Cohort Effect*

2.15.1 The influence of year of birth on mortality improvement rates has been the subject of much research in the past. The GAD produces national population projections incorporating projections of future mortality. Recent projections have been heavily influenced by trends linked to year of birth. For instance, the report describing the 1992 projection contained an analysis of the effect of year of birth on mortality improvement rates (GAD, 1995). The following is an extract from that report:

> "... higher than average rate of improvement is a special feature of generations born between 1925 and 1945 (which more detailed charts show to be centred on the generation born in 1931). It is not yet understood precisely why the members of the generation born about 1931 have been enjoying so much lower death rates throughout adult life than the preceding generation ..."

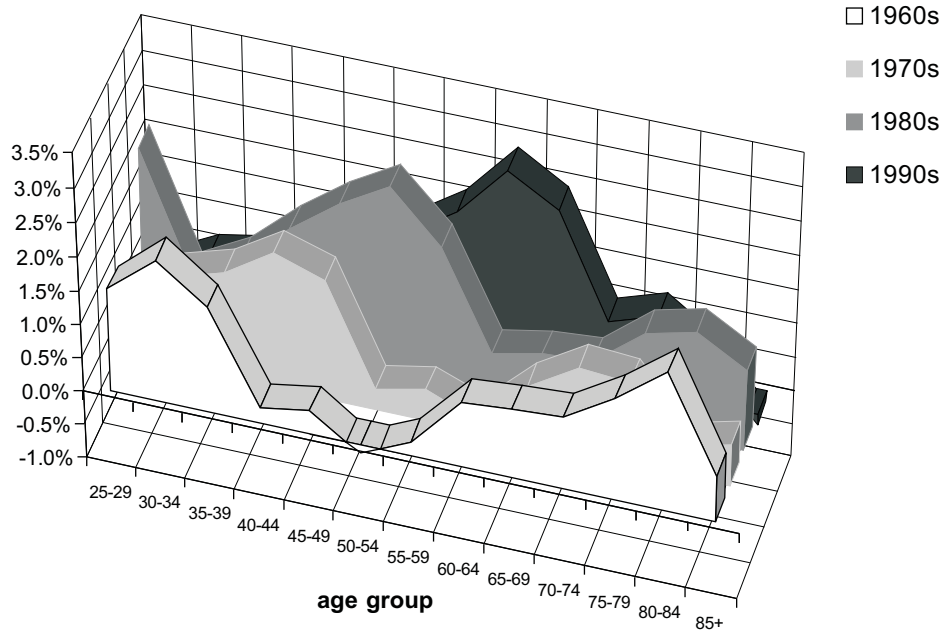
2.15.2 Figures 2.15a and 2.15b (and the underlying Tables 2.15a and 2.15b) illustrate this 'cohort effect' by showing the ages that have experienced the greatest improvements over the last four decades. Each of the blocks represents a decade and, for each decade, average annualised mortality improvement rates are shown for five-year age bands.

2.15.3 It can clearly be seen that the greatest mortality improvements in the 1960s were experienced by people then in their 30s, in the 1970s it was people then in their 40s, in the 1980s it was people then in their 50s and in the



Own figures — data source; O.N.S. (2003)

Figure 2.15a. Average annual rate of mortality improvement, England and Wales population, by age group and decade, males



Own figures — data source; O.N.S. (2003)

Figure 2.15b. Average annual rate of mortality improvement, England and Wales population, by age group and decade, females

Table 2.15a. Average annual rate of mortality improvement, England and Wales population, by age group and decade, males

Age group	1960s	1970s	1980s	1990s
25-29	**1.3%**	0.1%	0.4%	−1.0%
30-34	**1.5%**	1.5%	−0.6%	−0.9%
35-39	**1.5%**	**1.0%**	0.2%	1.0%
40-44	−0.2%	**2.2%**	2.2%	0.6%
45-49	−0.1%	**1.8%**	**2.4%**	1.1%
50-54	0.0%	0.6%	**3.2%**	2.5%
55-59	0.9%	1.1%	**3.1%**	**2.4%**
60-64	0.6%	0.9%	1.7%	**3.2%**
65-69	−0.0%	1.4%	1.8%	**3.1%**
70-74	−0.0%	1.1%	1.5%	1.9%
75-79	0.5%	0.4%	1.5%	2.0%
80-84	1.5%	−0.1%	1.4%	1.4%
85 and over	−0.2%	0.7%	1.2%	0.5%

Own figures — data source: O.N.S. (2003)

Table 2.15b. Average annual rate of mortality improvement, England and Wales population, by age group and decade, females

Age group	1960s	1970s	1980s	1990s
25-29	**1.5%**	0.6%	2.6%	0.2%
30-34	**2.1%**	1.3%	0.9%	0.6%
35-39	**1.6%**	**1.6%**	1.2%	0.8%
40-44	0.2%	**2.0%**	1.9%	0.4%
45-49	0.4%	**1.8%**	**2.4%**	1.0%
50-54	−0.1%	0.3%	**2.8%**	1.6%
55-59	0.2%	0.5%	**2.1%**	**2.1%**
60-64	1.0%	0.2%	0.6%	**2.8%**
65-69	1.0%	0.8%	0.8%	**2.4%**
70-74	1.1%	1.3%	0.8%	0.9%
75-79	1.5%	1.2%	1.4%	1.1%
80-84	2.1%	0.4%	1.6%	0.6%
85 and over	0.7%	0.6%	1.2%	−0.2%

Own figures — data source: O.N.S. (2003)

1990s it was people then in their 60s. This illustrates the 'cohort effect' centred on 1931.

2.15.4 A very similar cohort pattern can be seen for females. This has happened despite the fact that, as discussed in Section 2.6, certain causes of death (heart disease for example) have far more relative importance for men than women.

2.15.5 Figure 2.15c illustrates the 'cohort effect' in a different way, by notionally assigning rates of improvement to particular years of birth. For instance, the reduction in the mortality rate at age 50 between 1999 and 2000,

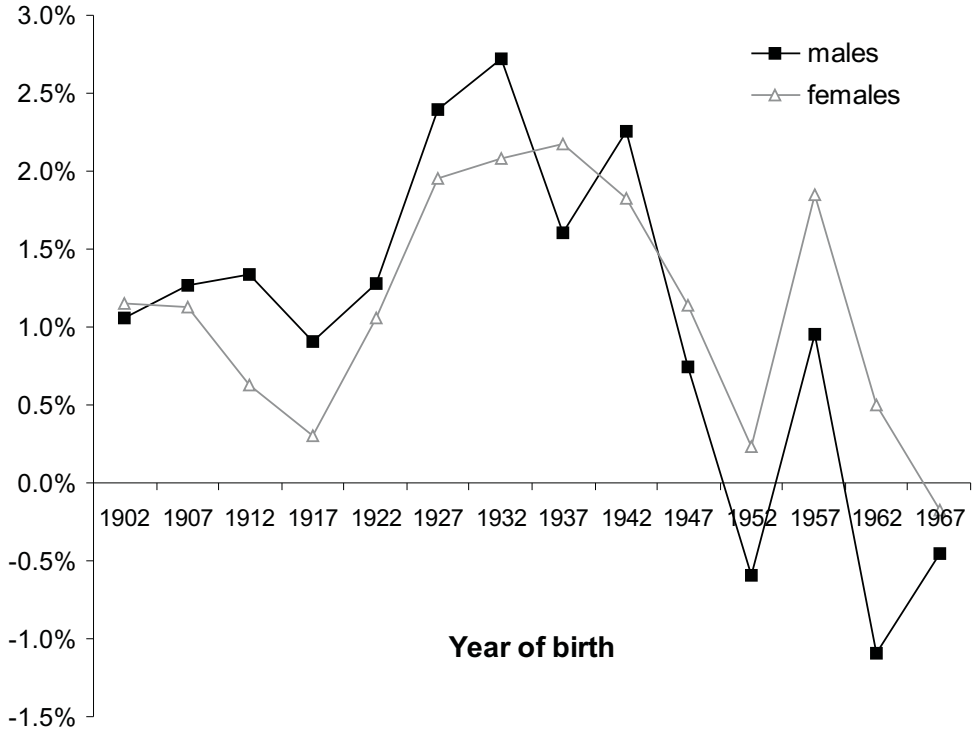


Figure 2.15c. Average annual rate of mortality improvement by year of birth, England and Wales population data from 1961 to 2001, ages of 20 to 89

is assigned to year of birth 1950. Average annual improvement rates for the period 1961 to 2001 have been calculated by year of birth (using data for ages 20 and above only). The data has actually been grouped into five year bands so the figure for '1902' (for instance) represents the average rate of improvement for years of birth from 1900 to 1904. The mass of high improvements for people born between 1925 and 1945 can clearly be seen for both males and females. For males, there appear to be two peaks within the 1925-1945 group. The first peak is centred on the early 1930s, while the second is centred on the early 1940s. Willets (2003) discusses the existence of these two 'sub-cohorts' in more detail.

2.15.6 There does appear to be evidence that some cohorts born after 1945 have experienced particularly poor mortality improvements. People born in the early 1950s and the 1960s have not fared well relative to people born in between these two periods. This feature could be more due to 'period-specific', rather than 'cohort-specific' influences, but it is certainly something to watch for in the future.

2.15.7 There are a number of possible explanations for why the cohort effect has occurred. These include:

— people in the 'healthy generation' (that is, those born between 1925 and 1945) have smoked fewer cigarettes than the previous generation;
— the 'healthy generation' were not as adversely affected as the previous generation by the depression of the 1930s;

— the 'healthy generation' were not involved in active service during World War II, whereas many people in the previous generation were;
— the 'healthy generation' benefited more from the introduction, in the late 1940s, of the state education system and the National Health Service; and
— the 'healthy generation' benefited most from the relatively healthy diet prevalent in the post-war years.

2.16 *The Cohort Effect for Individual Causes of Death*

2.16.1 In order to derive a clearer appreciation of the exact nature of the cohort effect, trends have also been analysed by cause of death. Tables 2.16a and 2.16b show improvement rates for mortality separately for four cause groups: circulatory disorders; cancer; respiratory disorders; and infectious diseases. Average annual rates of mortality improvement are shown for each decade, from the 1960s to the 1990s, subdivided here into 10-year age bands. In a similar manner to tables 2.15a and 2.15b, the age group(s) showing a local peak in the rate of mortality improvement in each decade are highlighted.

2.16.2 The pattern revealed by this analysis is striking. For circulatory disorders, cancer and respiratory disorders, and for both males and females, a clear cohort effect can be seen, in each case running along the two diagonals mainly representing years of birth 1925 to 1945. Even for infectious diseases a similar pattern is suggested although the overall picture is somewhat less clear. The conclusion is that people born in or around the 1930s have experienced consistently higher rates of improvement than those born into earlier or later generations, *in all the major health-related categories of death*.

2.16.3 This strongly suggests that the cohort effect is much more than mere coincidence (say a function of people born in the 1930s being of the right age, by chance, to benefit most from the improvements that have occurred in each decade). It appears to be a strongly rooted trend, which is highly likely to continue into the 21st century.

2.16.4 It is possible the cohort effects exhibited by each of the major cause-of-death groups may have different drivers and may be centred on slightly differing years of birth. The data presented here is insufficient to draw firm conclusions in this regard. However, a more detailed analysis of cause-specific trends by year of birth is contained in Willets (2003). This paper suggests that trends in smoking prevalence have been a key driver of the rapid improvements experienced by men and women born in the 1930s.

2.16.5 However, it also suggests that the second 'sub-cohort' of high improvement centred on the early 1940s has been caused by a different set of factors. Notably, the rapid rates of improvement in heart disease mortality experienced by people born in the 1940s are likely to have been driven by changes in the early life experience (e.g. diet) of those born in or around

Table 2.16a. Average annual rate of mortality improvement, England and Wales population, by age group and decade and category of death, males

Age band	1960s	1970s	1980s	1990s
Circulatory disorders				
30-39	−0.6%	**2.7%**	3.2%	2.4%
40-49	−3.5%	1.8%	**4.9%**	3.0%
50-59	−2.6%	0.3%	4.2%	**5.0%**
60-69	−2.3%	0.5%	2.6%	4.6%
70-79	−1.8%	1.0%	1.8%	3.2%
Cancer				
30-39	**1.0%**	0.9%	1.2%	2.6%
40-49	0.7%	**2.3%**	1.4%	1.7%
50-59	0.2%	0.7%	**1.9%**	2.0%
60-69	−0.7%	0.2%	0.4%	**2.6%**
70-79	−1.4%	−0.5%	−0.5%	1.6%
Respiratory disorders				
30-39	**2.3%**	5.3%	2.0%	0.5%
40-49	2.2%	**6.3%**	6.1%	−4.0%
50-59	2.4%	5.8%	**7.0%**	−1.7%
60-69	0.8%	4.9%	5.0%	**0.2%**
70-79	−1.1%	2.0%	5.5%	−1.6%
Infectious diseases				
30-39	**11.6%**	4.0%	−7.5%	4.7%
40-49	9.1%	**7.2%**	−2.3%	2.4%
50-59	8.7%	6.5%	**2.6%**	0.2%
60-69	8.1%	7.0%	1.6%	−1.1%
70-79	5.9%	5.4%	−0.2%	−3.2%

Own figures — data source: O.N.S. (2003)

World War II. This theory is very much in accordance with recent epidemiological research into the impact of early life experience on health in later life.

2.17 *The Cohort Effect for Insured Lives*

2.17.1 So far we have looked at how the cohort effect has affected population, rather than insured life, mortality experience. The differences between rates of mortality improvement for the general population and insurance data are discussed in depth in Section 2.23. However, figures showing evidence of a cohort effect in insured lives are briefly considered now.

2.17.2 Willets (1999) suggested that a similar generational effect could be seen in U.K. assured life experience. More substantial evidence for this was published in a C.M.I. Bureau working paper (2002) which investigated the possible existence and impact of such an effect. The C.M.I. Bureau working paper noted the existence of a similar trend, centred on a slightly

Table 2.16b. Average annual rate of mortality improvement, England and Wales population, by age group and decade and category of death, females

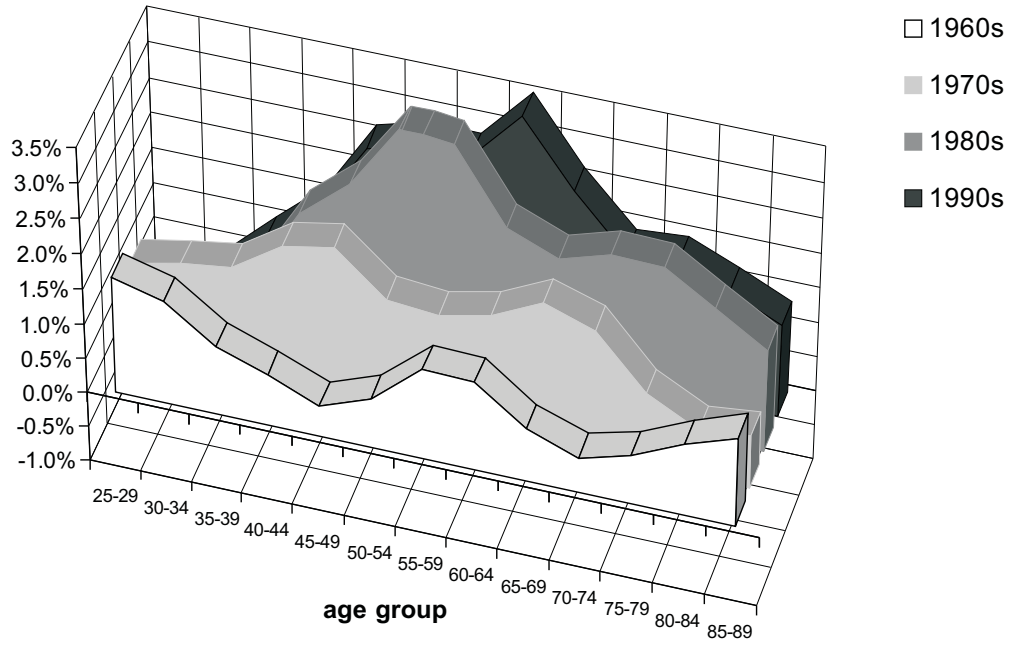
Age band	1960s	1970s	1980s	1990s
Circulatory disorders				
30-39	−0.2%	**3.3%**	3.5%	−0.2%
40-49	−2.4%	2.5%	**6.0%**	0.0%
50-59	−2.7%	1.1%	4.6%	4.1%
60-69	−1.8%	1.1%	2.5%	**4.7%**
70-79	−1.5%	1.8%	2.1%	3.2%
Cancer				
30-39	**1.5%**	0.7%	−0.1%	2.8%
40-49	−0.2%	**1.4%**	**1.1%**	2.1%
50-59	−1.1%	−0.4%	1.0%	**2.3%**
60-69	−0.5%	−1.0%	−0.9%	2.1%
70-79	0.0%	−0.6%	−1.5%	0.1%
Respiratory disorders				
30-39	**1.1%**	**6.3%**	4.9%	−5.4%
40-49	−0.6%	4.6%	**8.3%**	−3.6%
50-59	−1.6%	2.8%	4.9%	**−1.4%**
60-69	0.2%	2.3%	2.4%	−1.6%
70-79	−0.3%	1.1%	6.0%	−6.1%
Infectious diseases				
30-39	**11.2%**	**6.6%**	2.5%	−6.0%
40-49	5.4%	6.3%	**3.1%**	−4.6%
50-59	3.1%	3.9%	2.4%	**−1.2%**
60-69	5.3%	3.1%	−2.0%	−2.0%
70-79	5.2%	1.8%	−3.0%	−3.3%

Own figures — data source: O.N.S. (2003)

earlier generation. Data for male assured lives provided clear evidence of year of birth related effects in mortality improvement, with the cohort centred on births in 1926 the most pronounced.

2.17.3 The working paper also described an analysis of data for male life office pensioners, retiring at or after normal retirement age. A similar cohort effect was noted in this data set, with the peak improvements also occurring in the 1926 cohort.

2.17.4 These cohort trends were incorporated into future mortality projections outlined in the paper. The mortality projections assumed that the 'width' of the generation experiencing rapid improvement would reduce with time. For the period 1992 to 2000 the width was taken to be 33 years (i.e. those born between 1910 and 1942). It was then assumed to reduce linearly to one year (i.e. 1926) by the end of the 'cohort period.' The 'cohort period' was taken as being ten, 20 and 40 years for 'short cohort', 'medium cohort' and 'long cohort' projections respectively.



Own figures — data source; C.M.I. Bureau (2002)

Figure 2.17. Average annual rate of mortality improvement, assured lives, all durations, by age group and decade, males

2.17.5 Figure 2.17 (and the underlying Table 2.17) has been constructed using smoothed C.M.I. assured lives data. The smoothed data set was published alongside the working paper.

2.17.6 It can be seen that roughly the same pattern applies as for the general population, although there appears to be slightly less of a contrast

Table 2.17. Average annual rate of mortality improvement, assured lives duration 2 years and above, by age group and decade, males

Age group	1960s	1970s	1980s	1990s
25-29	**1.7%**	1.4%	−0.1%	0.3%
30-34	**1.5%**	1.5%	−0.1%	0.3%
35-39	**1.0%**	**1.6%**	0.9%	1.0%
40-44	0.7%	**2.1%**	2.0%	1.6%
45-49	0.4%	**2.2%**	**2.6%**	2.6%
50-54	0.7%	1.6%	**3.5%**	2.8%
55-59	1.3%	1.5%	**3.5%**	**2.8%**
60-64	1.3%	1.7%	2.4%	**3.5%**
65-69	0.8%	2.0%	2.2%	**2.6%**
70-74	0.5%	1.8%	2.4%	1.9%
75-79	0.7%	1.1%	2.3%	1.9%
80-84	1.0%	0.7%	1.9%	1.6%
85-89	1.3%	0.8%	1.5%	1.3%

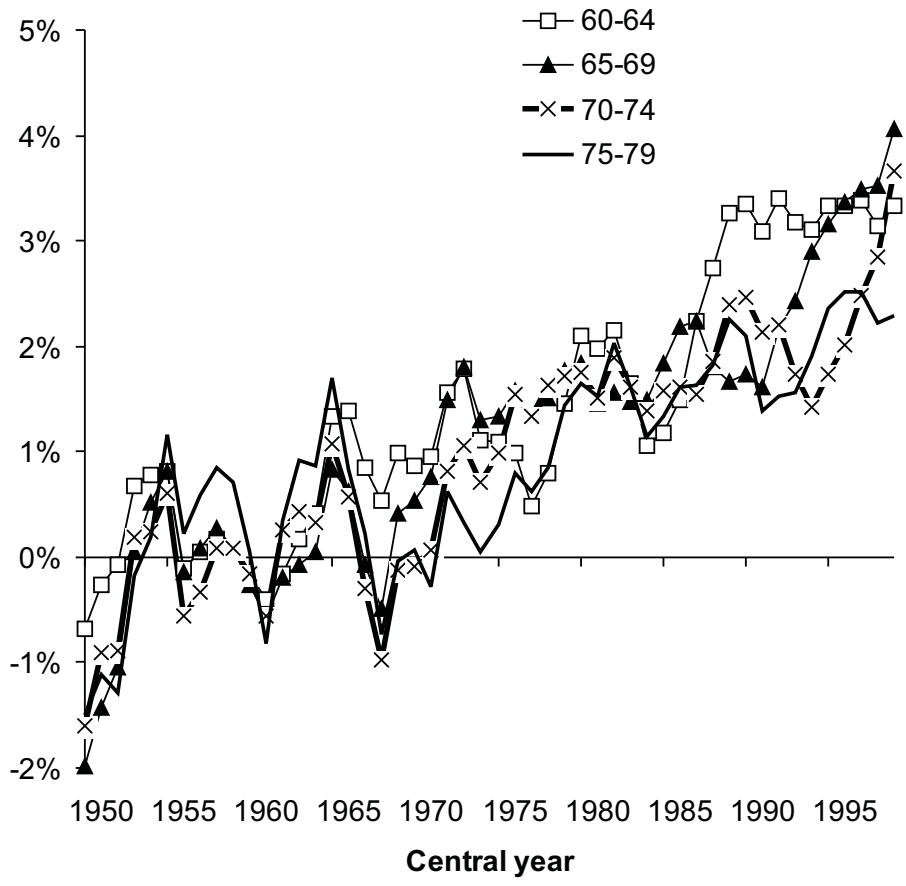
Own figures — data source: C.M.I. Bureau (2002)

between the improvements experienced by the 'healthy generation' and the generations born either side of it.

2.18 *The 'Ageing of Mortality Improvement'*

2.18.1 The idea of the 'ageing of mortality improvement' was briefly introduced in Section 2.3, where it was noted that the ages showing the greatest mortality improvements had moved upwards during the course of the 20th century. The concept was described by the American demographer John Wilmoth, who noted an 'ageing of mortality decline' in several countries around the world (Wilmoth, 1997).

2.18.2 There is some overlap between this concept and the cohort effect, since cohort trends automatically lead to mortality improvements moving up through the ages. However, there is a lot more to the 'ageing' concept than just a restatement of the cohort effect. This is shown by Figure 2.18a.



Own figures — data source; O.N.S. (2003)

Figure 2.18a. Average rate of mortality improvement, England and Wales population, seven-year rolling averages using data from 1946 to 2001, ages 60 to 79, males

2.18.3 Figure 2.18a illustrates how rates of mortality improvement for men in their 60s and 70s have accelerated in post-war England and Wales. Data for the whole period 1946 to 2001 are used, as the graph plots the average rate of annual improvement over rolling periods of seven years. The mortality experience of people born in the 'healthy generation' (roughly 1925 to 1945) will clearly have had some impact on the results in the most recent years, but only a relatively minor influence. In particular, experience for the 75 to 79 age-group is entirely based on experience for men born prior to 1925. Later in the paper, in Section 2.21, the equivalent figure for men in their 30s and 40s is given, which shows completely the opposite trend.

2.18.4 There are two aspects to the idea of the ageing of mortality improvement, namely:

— the ages showing the greatest rates of mortality improvement are increasing over time; and
— the pace at which mortality is improving at 'older ages' is accelerating over time (and the definition of 'old' is also moving upwards).

2.18.5 The second point can be illustrated by considering how the mortality experience of the very oldest people has improved. In an article in the O.N.S. publication *Population Trends*, Roger Thatcher (1999) described the 'explosion' in the number of centenarians in England and Wales. At the beginning of the 20th century there were roughly 100 people aged 100 and over. By the end of the century there were nearly 6,000. Thatcher contrasted this figure with a GAD projection which suggested that this number was likely to increase to 95,000 by the year 2066.

2.18.6 According to Thatcher, the largest single cause behind the rapid growth in the number of centenarians is the fall in mortality rates for people aged between 80 and 100. Some sample figures showing the proportion of 80-year-olds surviving to age 100 are given in Figure 2.18b.

2.18.7 Figure 2.18b would appear to counter the view that mortality rates are unlikely to improve significantly at very advanced ages. There is also evidence that mortality has also improved at ages in excess of 100 (Thatcher, 1999).

2.18.8 Interestingly, the rate at which mortality rates increase with age appears to tail off above the age of 100. Thatcher has derived figures, using an international database of old age mortality (the so-called Kannisto-Thatcher database), which indicate that mortality rates gradually approach something like a plateau — at a level below 50% — rather than tending exponentially towards 100%.

2.18.9 This finding is consistent with biological research involving large populations of creatures such as flies and worms and studies into the longevity of man-made machines, such as cars (Vaupel, 1997). The common thread is that mortality curves are found to decelerate at older ages.

2.18.10 The research into mortality at very old ages, combined with the

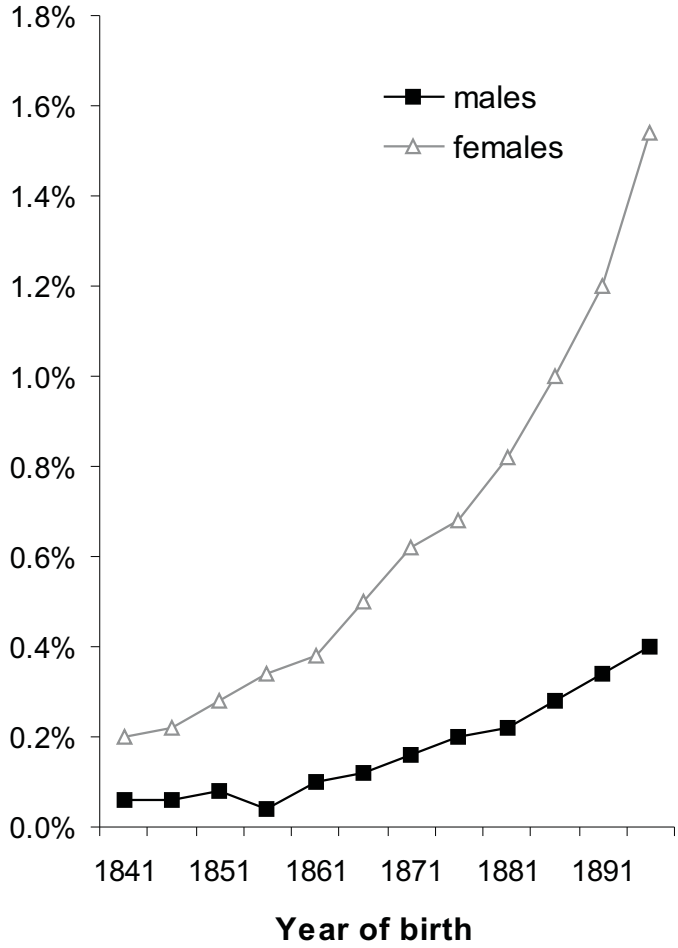


Figure 2.18b. Proportion of 80-year-olds surviving to age 100, England and Wales, approximate values based on Figure 4 in Thatcher (1999)

observed ageing of mortality improvement, has led actuaries and demographers to question some commonly-held views. Theories suggesting a maximum human lifespan that cannot be breached, or a limiting distribution below which mortality rates cannot fall, probably appeared more credible before the emergence of the improvements in elderly mortality witnessed in recent decades.

2.18.11 This topic will be discussed further in Section 3 — which considers data from other countries around the world.

2.19 *The Impact of Cigarette Smoking on Recent Mortality Trends*

2.19.1 The causes of death responsible for the greatest contributions to recent mortality improvements are strongly linked to cigarette smoking behaviour. These causes include heart disease and lung cancer. Another way of putting this is to say that *part* of the mortality improvements in recent years have been *the result* of changes in smoking behaviour.

2.19.2 An obvious question to ask is: "What part of the improvements was due to changes in smoking behaviour, and what part was due to other factors?" In order to answer this question a simple model was constructed to remove the effect of changes in smoking behaviour from total mortality improvements. It was assumed that aggregate mortality rates can be expressed as:

% that are smokers × never-smoker mortality rate × smoker adjustment +
% that are ex-smokers × never-smoker mortality rate × ex-smoker adjustment +
% that have never smoked × never-smoker mortality rate.

2.19.3 The 'smoker adjustment' is the assumed differential between never-smoker and current-smoker mortality rates. The 'ex-smoker adjustment' is the assumed differential between never-smoker and ex-smoker mortality rates. In fact, various ex-smoker differentials are used which depend on the period elapsed since giving-up cigarettes. All the differentials are assumed to remain constant over time.

2.19.4 The model was used to produce male mortality rates for the 40 to 49 and 50 to 59 age-groups standardised by smoking prevalence.

2.19.5 In order to construct the model the following data were collected:

— changes in mortality rates by age over time;
— changes in cigarette smoking prevalence by age over time; and
— differentials between smoker, ex-smoker and non-smoker mortality.

2.19.6 The figures on smoking prevalence were taken from the General Household Survey (O.N.S. 2002). Some sample figures are shown in Table 2.19a.

2.19.7 Table 2.19a shows significant reductions in the prevalence of cigarette smoking during the 1980s, with evidence of stabilisation in the 1990s (at least for the 35 to 59 age-group).

2.19.8 The model makes assumptions about the proportion of smokers in each year *and* the proportion of ex-smokers, split by duration since

Table 2.19a. Prevalence of cigarette smoking in Great Britain by age and calendar year, males, percentages

Age group	Year												
	1974	...	1980	1982	1984	1986	1988	1990	1992	1994	1996	1998	2000
20-24	52	...	44	41	40	41	37	38	39	40	43	41	35
25-34	56	...	47	40	40	37	37	36	34	34	38	38	39
35-49	55	...	45	40	39	37	37	34	32	31	30	33	31
50-59	53	...	47	42	39	35	33	28	28	27	28	28	27
60+	44	...	36	33	30	29	26	24	21	18	18	16	16

Source: O.N.S. (2002)

Table 2.19b. Mortality differentials used in the model

Smoker category		Differential (mortality relative to never-smokers)
Current smoker		300%
Ex-smoker	1 year since quitting	265%
Ex-smoker	2-5 years since quitting	185%
Ex-smoker	6 years since quitting	175%
Ex-smoker	7 years since quitting	165%
Ex-smoker	8 years since quitting	150%
Ex-smoker	9 years since quitting	140%
Ex-smoker	10-11 years since quitting	130%
Ex-smoker	12-15 years since quitting	120%
Ex-smoker	16 years plus since quitting	100%
Never smoker		100%

quitting. The various ex-smoker proportions were derived by assuming (for simplicity) that the transition from smoker to non-smoker is a one-way process, that is people who quit cigarettes do not return to smoking.

2.19.9 The mortality differentials used in the model were derived from two sources: a long-term study into the mortality of male British doctors (Doll *et al.*, 1994) and American figures on the relationship between smoking cessation and improved mortality (Housholder, 1998).

2.19.10 The current smoker ratio is higher than figures typically seen for insured lives. There are a number of reasons for this, which include:

— the non-disclosure of true smoking status by a proportion of current smokers when applying for insurance;
— the fact that some smokers will give up smoking during the term of insurance policies;
— conversely, some non-smokers will become smokers — especially ex-smokers; and
— ex-smokers who have not smoked for 12 months are generally classified as non-smokers for insurance purposes.

2.19.11 The results of applying the model to male mortality improvements over 1980 to 2000, for ages 40 to 59, are given in Table 2.19c.

Table 2.19c. Mortality improvement rates with the effect of changes in smoking prevalence stripped out, England and Wales population, males

Age group	Annual rate of mortality improvement			
	Before adjustment		After adjustment	
	1980s	1990s	1980s	1990s
40-49	2.3%	0.8%	0.9%	–0.3%
50-59	3.1%	2.7%	1.6%	1.8%

Figures were only produced for males in this age-group, as the smoker differentials were taken from a study that did not include data for females nor for many males at older or younger ages.

2.19.12 The methodology used to produce the above figures has a number of obvious limitations, for instance:

— the mortality differentials are based on a combination of United States figures and U.K. data from the period 1971-1991;
— the differentials are assumed to remain constant over time; and
— no allowance is made for the correlation between smoking and other harmful activities (for example, excess drinking or poor diet).

2.19.13 Despite these limitations, the adjusted mortality improvement rates are useful as they indicate that a significant proportion of the recent high level of improvements has been due to changes in smoking behaviour. However, they also show that a significant proportion has been due to other factors. This is consistent with the findings of other studies. For instance, it is believed that the change in cigarette consumption has been responsible for between a quarter and a third of the reduction in heart disease mortality since the 1970s (National Heart Forum, 1999).

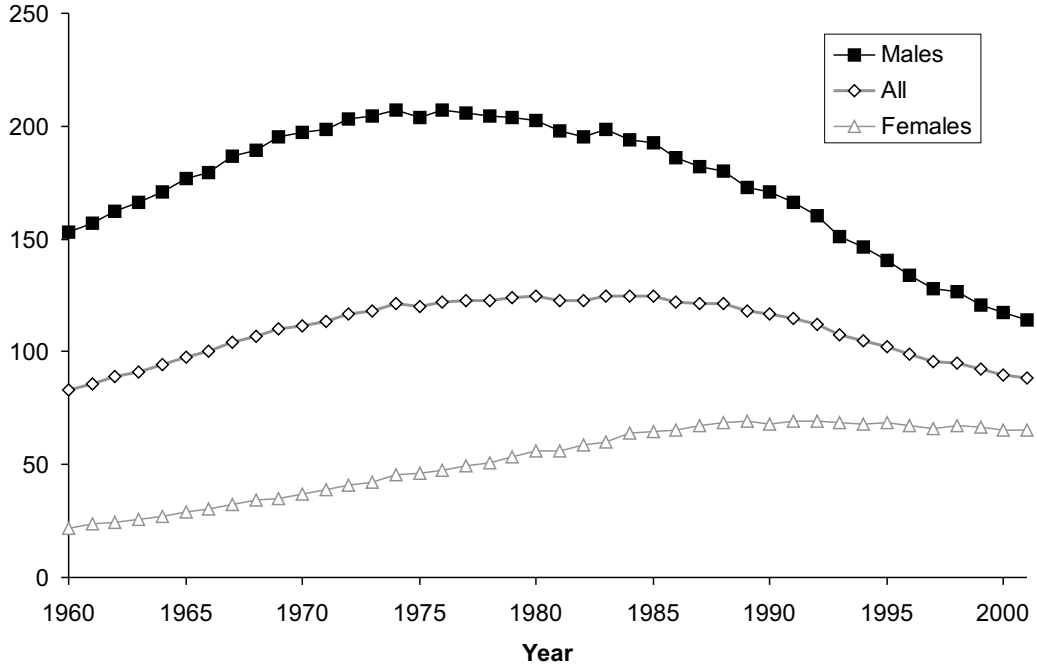
2.20 *The Impact of Cigarette Smoking on Longer-Term Mortality Trends*

2.20.1 The *recent* impact of changes in cigarette smoking prevalence is relatively clear-cut for middle-aged men. However, the effect over longer periods of time, and over wider age groups, is more difficult to judge. For males, cigarette consumption in Britain climbed steadily during the period 1900 to 1940, stayed constant from 1940 to 1960, fell steadily during the 1960s and 1970s and has showed signs of stabilisation in the late 1980s and 1990s (O.N.S. 2002).

2.20.2 Cigarette consumption for females has historically been much lower than for males. The peak rate of consumption occurred around 1960 after slow, but steady, growth from the early part of the century. In 1960 the average cigarette consumption of females was still less than half of that of males. During the 1960s and 1970s, consumption fell slightly, and then stabilised in the 1980s. By the end of the 1990s, rates of cigarette consumption and prevalence were broadly similar for males and females.

2.20.3 Taken on their own, these figures might suggest that the average improvement figures for the last four decades (that is, the figures given in Table 2.3) have been boosted by smoking-related improvements. However, a study of trends in lung cancer mortality, a cause of death very closely associated with cigarette smoking, shows the interaction over time, of changing patterns of cigarette consumption and mortality, to be far more complex.

2.20.4 Figure 2.20a shows that age-standardised rates of lung cancer mortality rose in the 1960s, peaked in the 1970s and fell throughout the 1980s



Own figures — data source: O.N.S. (2003)

Figure 2.20a. Age-standardised lung cancer mortality rates, England and Wales population, aged 30 plus

and 1990s for males. Despite falling prevalence of cigarette smoking, lung cancer mortality rates are actually *higher* now than in the early 1960s for women aged 50 and over and for men above the age of 75 (O.N.S. 2003).

2.20.5 Whilst the impact of cigarette smoking on other causes of mortality may differ in magnitude and timing, it seems likely the broad pattern shown in Figure 2.20a will be reflected in overall mortality trends.

2.20.6 Taking all adult ages together, patterns of male smoking behaviour resulted in very low rates of mortality improvement early in the period, followed by much greater improvements in the recent years. The overall effect is difficult to quantify. However, changes in smoking behaviour may not have *significantly* distorted *average* all-age mortality improvement rates over the whole period in question.

2.20.7 The adverse impact of cigarette smoking on an individual increases substantially with the *duration* for which that person has been smoking. Further, given the addictive nature of nicotine and that the majority of smokers take up the habit in early adulthood, the impact is also strongly correlated with age. This suggests that we may see cohort patterns within the lung cancer mortality data, with a key driver being the prevalence of cigarette smoking as each generation passed through their 20s.

2.20.8 Indeed this is the case:

— for males, lung cancer mortality rates have peaked and are falling, but

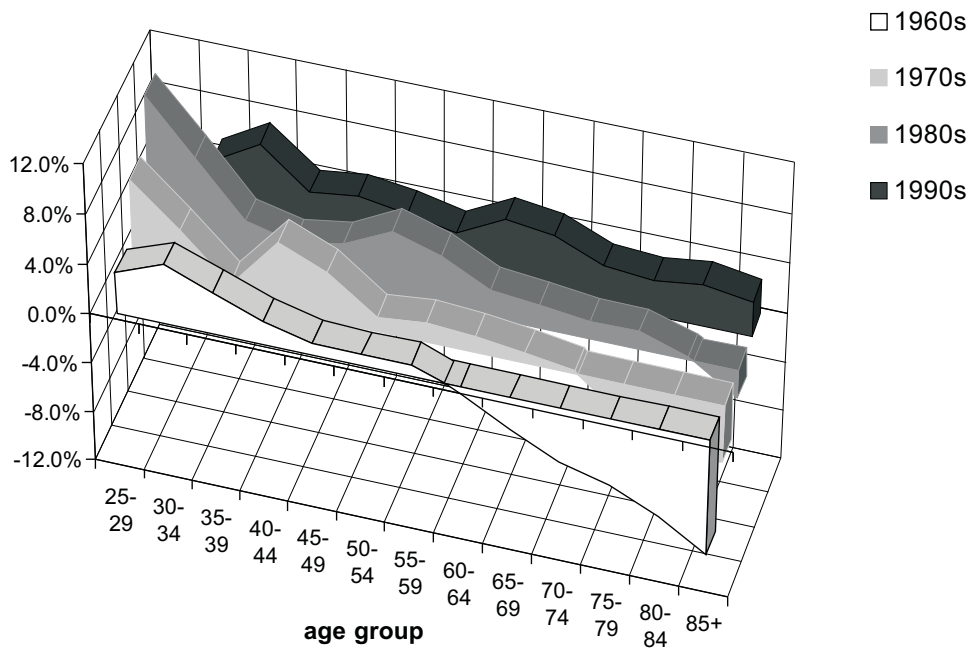
the peak has been later for each successive age group, each time relating to the cohort of men born in 1900-1905; and

— for females, the peak has also been later for each successive age group, but each time relating to the cohort of women born in 1925-1930; rates are therefore still rising for ages 75 and over.

2.20.9 Figures 2.20b and 2.20c (and the underlying Tables 2.20b and 2.20c) are akin to Figures 2.15a and 2.15b, but this time reflect the pattern of impact of cigarette smoking by showing changes in lung cancer mortality only.

2.20.10 Figures 2.20b and 2.20c show that the cohort of men and women born in the early 1930s has benefited from significantly lower lung cancer mortality than the preceding generation. Further detailed modeling work confirms that the observed pattern of changes in lung cancer mortality can be very largely explained just by reference to changing patterns of cigarette consumption. It seems, therefore, that the impact of changes in smoking habits over time has been a significant contributing factor to the overall cohort effect discussed in Section 2.15.

2.20.11 The figures also show other significant cohorts. Men born in the late 1940s and the 1950s are benefiting from particularly rapid reductions in



Own figures — data source: O.N.S. (2003)

Figure 2.20b. Average annual change in lung cancer mortality rates, England and Wales population, males, by decade and age group

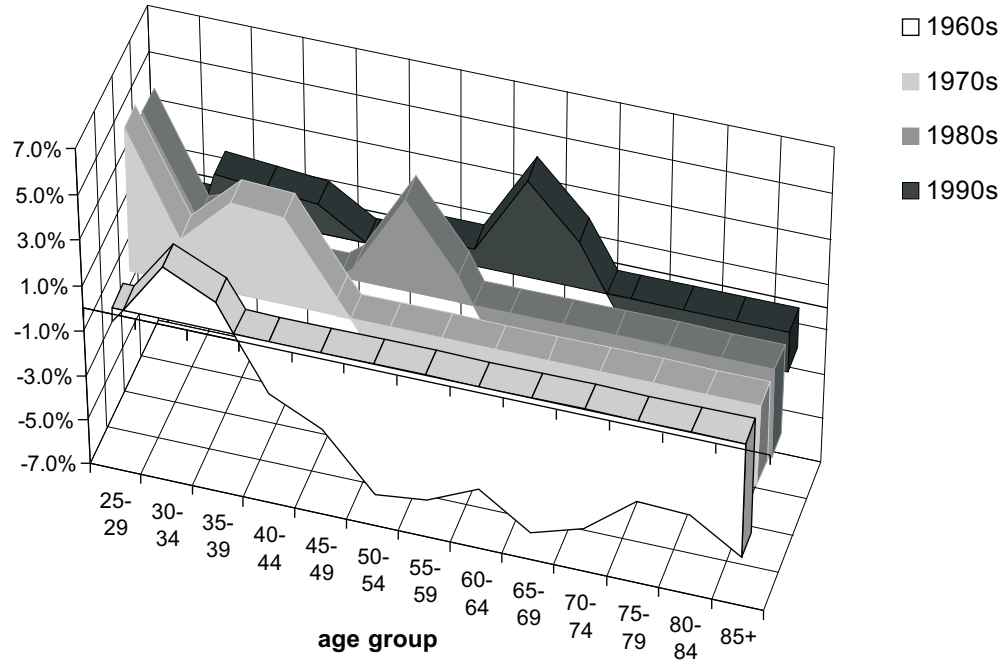


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Figure 2.20c. Average annual change in lung cancer mortality rates, England and Wales population, females, by decade and age group

Table 2.20b. Average annual change in lung cancer mortality rates England and Wales population, males, split by decade and age group

Age group	1960s	1970s	1980s	1990s
25-29	**3.3%**	8.0%	12.3%	−4.4%
30-34	**4.8%**	4.8%	7.9%	5.0%
35-39	**3.4%**	**1.3%**	3.7%	7.1%
40-44	1.9%	**5.5%**	2.8%	4.1%
45-49	1.0%	**3.9%**	**3.5%**	4.6%
50-54	0.9%	1.2%	**5.3%**	4.1%
55-59	1.0%	1.6%	**4.2%**	**3.3%**
60-64	−0.5%	1.3%	2.3%	**5.2%**
65-69	−2.6%	0.8%	1.9%	**4.7%**
70-74	−4.4%	0.2%	1.4%	3.1%
75-79	−5.3%	−2.8%	1.5%	2.8%
80-84	−7.1%	−5.8%	0.3%	3.3%
85 and over	−9.4%	−4.7%	−2.3%	2.8%

Own figures — data source: O.N.S. (2003)

lung cancer mortality compared to those born in the late 1930s/early 1940s. The cohort of men born around 1900 has been hardest hit by the effects of smoking. Mortality improvements for this cohort have been significantly dampened downwards by increased deaths from smoking-related causes. A similar observation applies to the cohorts of women born before 1925.

Table 2.20c. Average annual change in lung cancer mortality rates, England and Wales population, females, split by decade and age group

Age group	1960s	1970s	1980s	1990s
25-29	**−0.6%**	6.3%	5.4%	−5.5%
30-34	**2.3%**	1.9%	1.2%	1.6%
35-39	**1.2%**	**4.0%**	2.0%	1.4%
40-44	−2.3%	**3.8%**	−1.2%	1.2%
45-49	−3.4%	**0.8%**	**0.5%**	−0.1%
50-54	−5.9%	−2.4%	**4.0%**	0.7%
55-59	−5.6%	−3.4%	**1.1%**	**0.7%**
60-64	4.6%	−5.0%	−2.8%	**4.2%**
65-69	−6.1%	−5.0%	−3.2%	**2.0%**
70-74	−5.3%	−4.4%	−4.3%	−1.7%
75-79	−3.6%	−4.7%	−4.5%	−1.9%
80-84	−3.7%	−4.5%	−4.5%	−1.9%
85 and over	−5.1%	−3.6%	−4.0%	−1.8%

Own figures — data source: O.N.S. (2003)

2.21 *Increased Uncertainty for Young Adults*

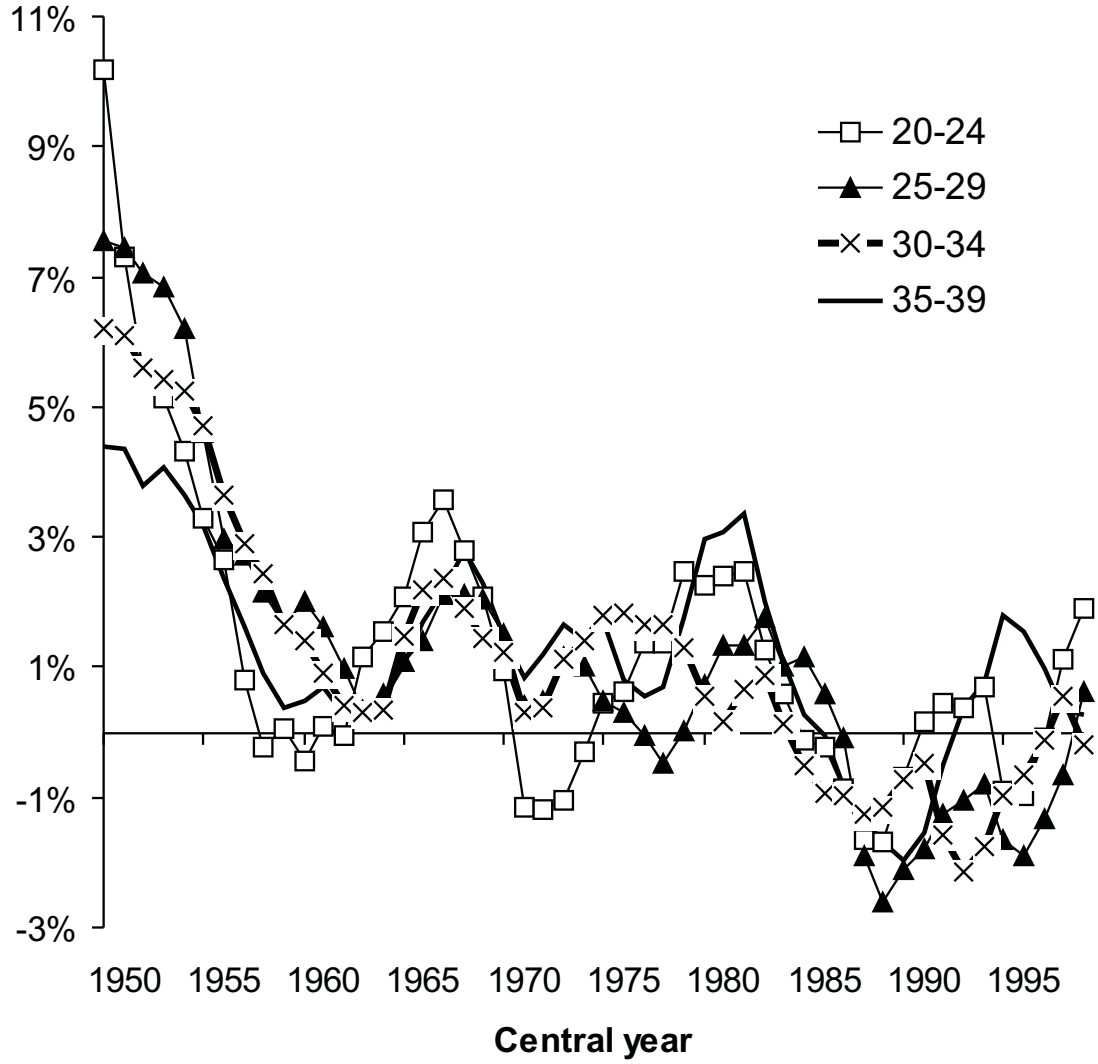
2.21.1 During the 1980s and 1990s, mortality rates increased for a number of causes that are relatively common in younger adults: these included AIDS, drug and alcohol abuse, liver disease, violence and accidental death. These deteriorations in experience have offset improvements in health-related causes of death.

2.21.2 In fact, mortality improvements for young men have been on the decline since the end of the Second World War. Figure 2.21 is the equivalent of Figure 2.18a which showed how improvement rates for men in their 60s and 70s have steadily increased over time. The trend shown in Figure 2.21 shows exactly the opposite pattern for men in their 20s and 30s, with rates of mortality improvement decelerating over time. Data for the whole period 1946 to 2001 are used as the graph plots the average rate of annual improvement over rolling periods of seven years.

2.21.3 Not only have aggregate improvement rates fallen, but many individual causes have shown deteriorations in experience. At younger ages there is a greater range of causes of death which can have a material impact on trends. It seems clear that the future course of mortality rates for younger adults is subject to considerable uncertainty.

2.22 *Widening Social Class Differentials*

2.22.1 In recent decades population data have shown widening mortality differentials by socio-economic class — that is, the mortality of the better off classes has improved more quickly. Figures 2.22a and 2.22b illustrate how differentials in life expectancy by socio-economic class have changed over the last three decades.

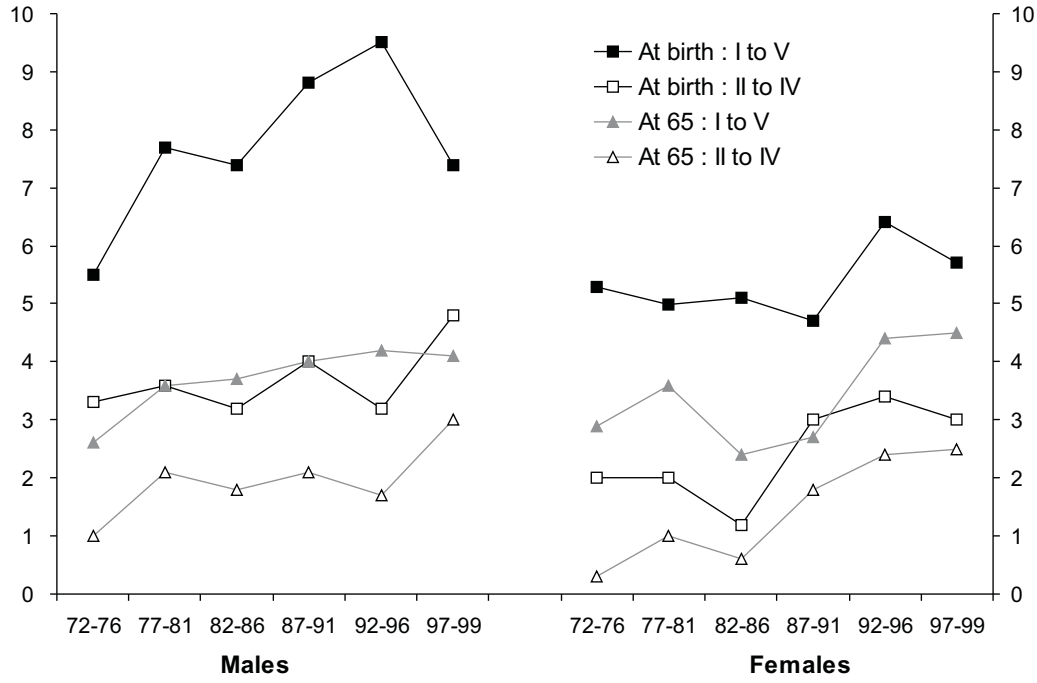


Own figures — data source; O.N.S. (2003)

Figure 2.21. Average rate of mortality improvement, England and Wales population, seven-year rolling averages using data from 1946 to 2001, ages 20 to 39, males

2.22.2 Differentials have actually grown fairly steadily throughout the post-war years (Department of Health, 1999). This appears to rule out one possible explanation for the cohort effect — namely that narrowing social class differentials could have caused mortality to improve rapidly for the generation born between 1925 and 1945. Instead, it suggests that the cohort effect may be seen *more strongly* in experience weighted towards higher socio-economic classes, such as the mortality of annuitants.

2.22.3 One reason for the existence of mortality differentials by socio-economic class is that cigarette smoking varies significantly according to social class. In 1997-99 the standardised mortality ratios (SMRs) for males aged 20-64 were 61% in Class I (professionals) and 157% in Class V



Source: Donkin *et al.* (2002)

Figure 2.22a. Differentials in life expectancy by socio-economic class, England and Wales 1972-1999

Source: Donkin *et al.* (2002)

Figure 2.22b. Differentials in life expectancy at age 65 between manual and non-manual socio-economic classes, England and Wales 1972-1999

Table 2.22a. SMRs for Classes I and V by cause of death, males in England and Wales aged 20-64, 1991-93

Cause	SMR for Class I	SMR for Class V
Heart disease	63%	182%
Stroke	70%	219%
Lung cancer	45%	206%
Skin cancer	136%	100%
Accidental	54%	226%
Suicide	55%	215%
All causes	66%	189%

Data source: O.N.S. (1997b)

(unskilled workers) (Donkin *et al.*, 2002). However, the General Household Survey (O.N.S., 2002) showed that smoking prevalence in these groups in 1998 was 15% and 45% respectively. If the mortality of smokers is assumed to be 300% of that of non-smokers, then differences in smoking prevalence explain 40% of the differential in SMRs. This suggests smoking is not the only factor behind socio-economic class differentials.

2.22.4 Table 2.22a shows that the mortality differentials by socio-economic class apply to many different causes of death, some of which are clearly not strongly related to smoking.

2.22.5 The framework most commonly used in discussing explanations for socio-economic mortality differentials was presented in the 'Black Report' (Townsend & Davidson, 1982). Three main types of explanation were outlined: selection explanations (e.g. unheathly people are less likely to be employed in highly skilled occupations); materialist explanations (e.g. poorer quality housing conditions cause health problems for the less skilled/ lower paid) and cultural explanations (e.g. differences in cigarette smoking prevalence and diet). Other hypotheses include Marmot's theory (e.g. Marmot *et al.*, 1997) that differentials are partly due to work related stress and in particular lack of control at work.

2.22.6 It is difficult to draw firm conclusions about the precise causes of class differentials. In an analysis of differentials in a range of developed countries Valkonen (2001) came to the conclusion that:

> "... explanations are likely to be different for different causes of death and in different countries and time periods. No universally valid explanations exist."

2.22.7 Having said that, an analysis of trends in mortality by social class for separate causes of death suggests that differing improvements in heart disease mortality have played a major role in widening differentials. Table 2.22b shows the components of average annual mortality improvements for males between the early 1970s and early 1990s split by socio-economic class.

Table 2.22b. Components of average annual mortality improvements rates 1970-72 to 1991-93, England and Wales population, males aged 20-64

Class	Heart disease	Lung cancer	Stroke	Accidents	Suicide	Other	All cause
I	1.4%	0.3%	0.3%	0.1%	0.0%	0.6%	2.7%
II	1.2%	0.3%	0.3%	0.1%	0.0%	0.7%	2.6%
IIIN	1.0%	0.3%	0.2%	0.1%	0.0%	0.4%	1.9%
IIIM	0.6%	0.3%	0.2%	0.1%	−0.1%	0.5%	1.5%
IV	0.6%	0.3%	0.2%	0.1%	0.0%	0.6%	1.8%
V	0.0%	0.2%	0.1%	0.1%	−0.1%	0.2%	0.5%
Total	0.8%	0.3%	0.2%	0.1%	−0.1%	0.6%	1.9%

Own figures — data source: O.N.S. (1997b)

2.22.8 In fact, in Europe as a whole, recent mortality improvement has been largely due to a decline in deaths from heart disease. Heart disease mortality is now much higher in lower socio-economic class groups than in other groups. However, this was not always the case. Heart disease mortality social class differentials began to emerge in Northern and Western Europe in the 1960s and 1970s and this has been the single greatest factor behind widening class differentials across Europe (Valkonen, 2001).

2.23 *Rates of Mortality Improvement for Pensioners and Assured Lives*

2.23.1 As socio-economic class mortality differentials have widened over time, you would expect — other things being equal — improvements for pensioners and assured lives to have been greater than in the general population. This statement is based on the fact that the socio-economic class mix of these groups is higher than the population average. Of course, there are distorting factors, such as changes in underwriting practice and the changing prevalence of life assurance or pension provision in different socio-economic groups. However, the experience for males has certainly backed up the general expectation.

2.23.2 Smoothed mortality rates from the C.M.I. assured lives investigation were made available as part of the recent C.M.I. Bureau study into cohort trends (C.M.I. Bureau, 2002). These data were also used in Section 2.17. Average annual rates of improvement for each individual age were derived using these data (using log linear regression to fit trend line) and compared with equivalent rates for the population of England and Wales. The result of this comparison is illustrated by Figure 2.23a. It can be seen that rates of improvement have been higher in the C.M.I. dataset at all ages. In fact, the differential has been remarkably similar for different ages, as illustrated by Figures 2.23b and 2.23c.

2.23.3 Figures 2.23a to 2.23c show that improvements for male assured lives have been roughly 0.4% to 0.5% p.a. higher (25% to 30%

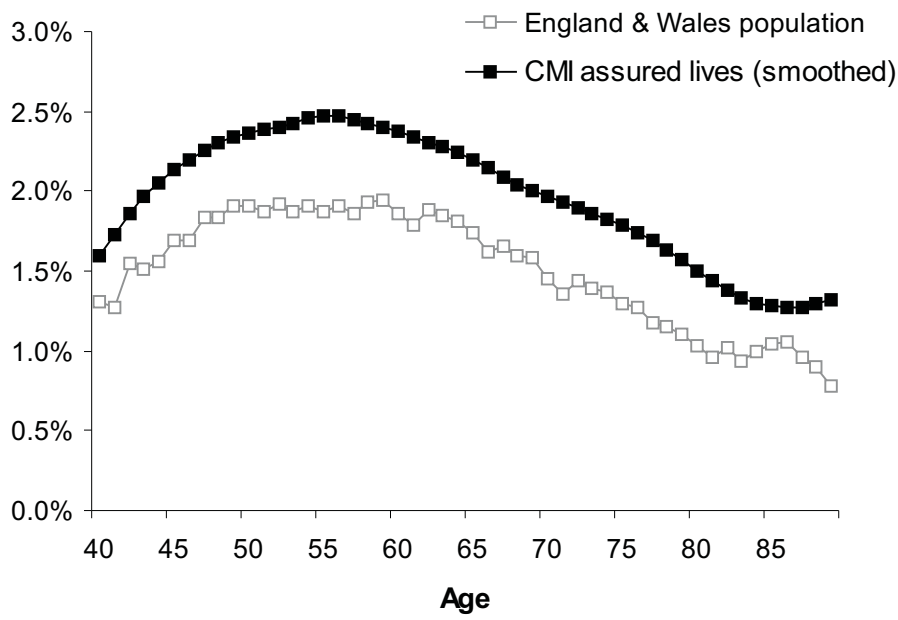


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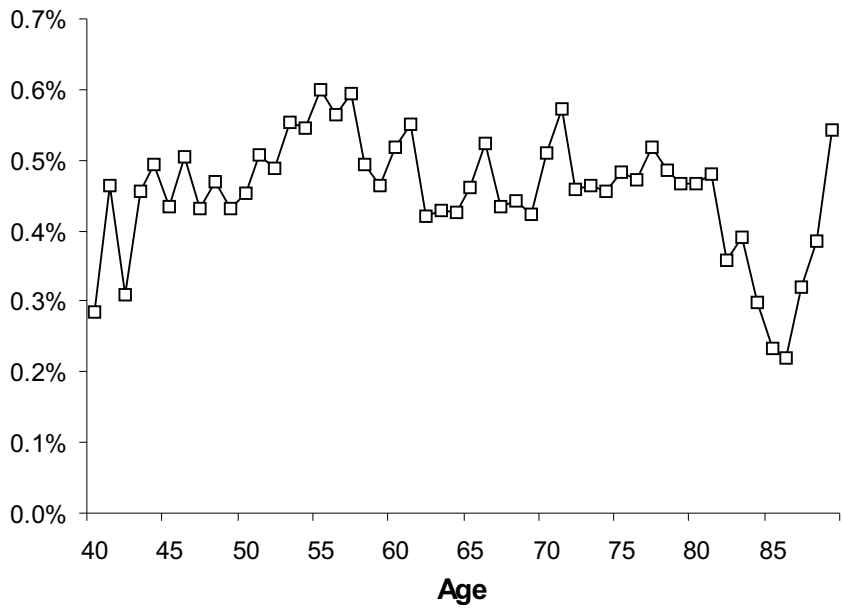
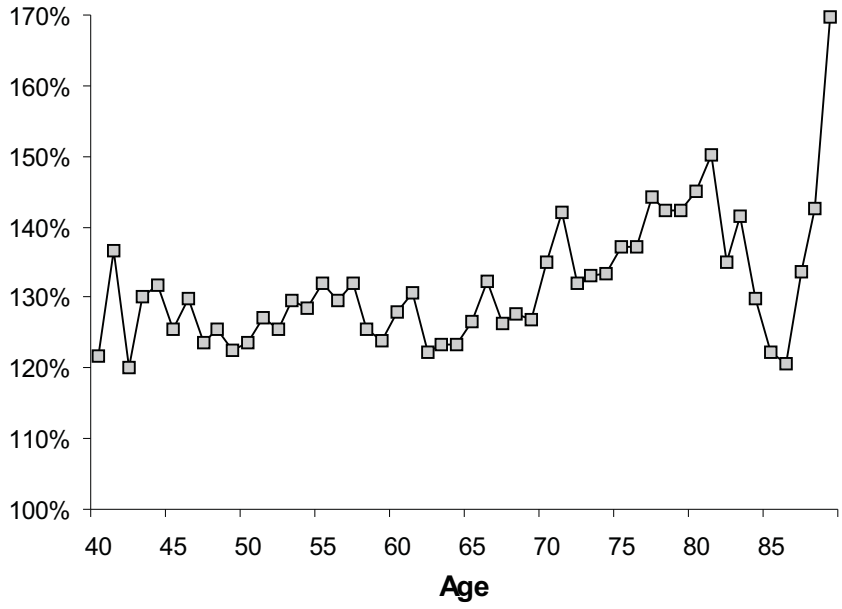
Figure 2.23a. Average annual rate of mortality improvement by age for males in the population of England and Wales and in the C.M.I. smoothed dataset for male assured lives 1961-99



Own figures: data source: C.M.I. Bureau (2002), GAD

Figure 2.23b. Difference between the average annual rate of mortality improvement by age for males in the C.M.I. smoothed dataset for assured lives and males in the population of England and Wales 1961-99



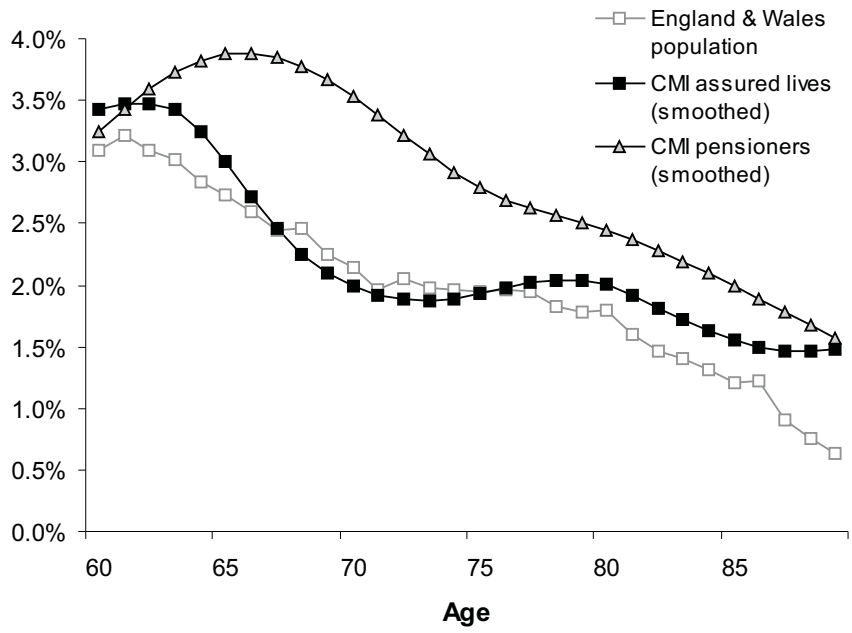
Own figures: data source: C.M.I. Bureau (2002), GAD

Figure 2.23c. Ratios of the average annual rate of mortality improvement by age for males in the C.M.I. smoothed dataset for assured lives to males in the population of England and Wales 1961-99

greater as a proportion) than rates for the population of England and Wales.

2.23.4 Figure 2.23d shows how improvement rates at older ages have compared in the more recent past. Improvements for life office pensioners have also been derived using the C.M.I. Bureau's smoothed dataset. Over the period 1983-1999, improvements experienced by assured lives have been close to those for the general population, whereas improvements for male pensioners have been considerably higher (more than 50% higher at most ages). It is also worth noting that the C.M.I. smoothed rates are based on lives-weighted experience. Historic improvements based on amounts-weighted data have shown even higher rates of change (Willets, 1999).

2.23.5 Improvement rates for women in the general population, women in the insured population and female pensioners have all been of a similar magnitude and shape in recent years — see Willets (1999) for more detail. The lack of a differential between population and insurance improvements for females may be due to growth in the exposed to risk for the female assured life and pensioners investigations. Specifically, the growth in the exposure may have been caused by more widespread distribution of products throughout the population, resulting in a change in the socio-economic class mix of assured lives and pensioners.



Own figures: data source: C.M.I. Bureau (2002), GAD

Figure 2.23d. Average rate of mortality improvement by age for males in the population of England and Wales and in the C.M.I. Bureau's 'smoothed' datasets 1983-99

3. International Experience

3.1 *Introduction*

The purpose of this Section of the paper is to see whether an understanding of international mortality experience will aid our predictions of future U.K. change. Present-day mortality rates, life expectancies and historic improvement rates in the U.K. are compared with other countries. International demographic research is also discussed.

3.2 *The Shape of the U.K. Mortality Curve*

3.2.1 Population mortality rates for the U.K. have a distinctive age 'shape' relative to similar countries around the world. Rates for the U.K. are comparatively low for younger adults, but increase steadily — in relative terms — with advancing age. Relative rates reach their peak in the 65 to 75 age range. This is illustrated by Figure 3.2a, which compares U.K. rates with an 'average' table constructed from tables produced in a range of different developed countries. Specifically, tables from the following countries were used in deriving the international 'average': Australia; Austria; Belgium; Canada; Denmark; Finland; France; Germany; Greece; Ireland; Israel; Italy; Japan; Netherlands; New Zealand; Norway; Portugal; Singapore; Spain; Sweden; Switzerland; U.K.; and the United States of America.

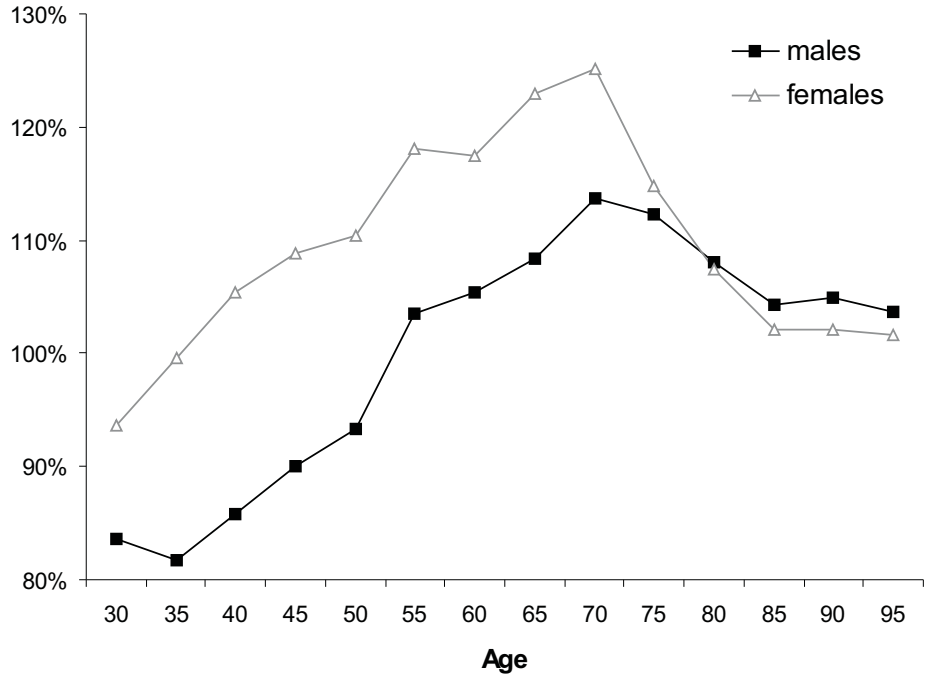


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Figure 3.2a. U.K. mortality rates by age relative to an international 'average'

3.2.2 Figure 3.2a shows that U.K. mortality rates are high — relative to similar countries — for males aged between 60 and 80 and females aged between 40 and 80. Over the age of 85 rates in the various tables converge to a much greater extent.

3.2.3 The difference between U.K. and U.S. mortality is interesting. As well as showing results for these two countries, Figures 3.2b and 3.2c also give the minimum and maximum rate (from the sample countries used in Figure 3.2a) at each age. There is clearly a wide range of experiences for countries that might be expected to be similar. Relative mortality for the U.S.A. is almost a mirror image of that for the U.K.: much higher at younger ages and lower from about age 70.

3.2.4 Figures 3.2b and 3.2c show that there is a 'narrowing funnel of variation', with the proportions for different countries converging with increasing age (the absolute rates also converge above age 85).

3.2.5 It is also apparent that for older adults the U.K.'s mortality rates are far above the minimum observed. For older males the country with lowest rate varies with age. It is Sweden at age 60, Switzerland at 65, Japan at 70 and at ages 75 and 80 it is France. For women of all ages between 60 and 80 Japanese mortality rates are lowest.

3.2.6 Table 3.2 shows that the mortality rate for a male aged 70 is almost 50% higher in the U.K. than the country with the lowest rate at this

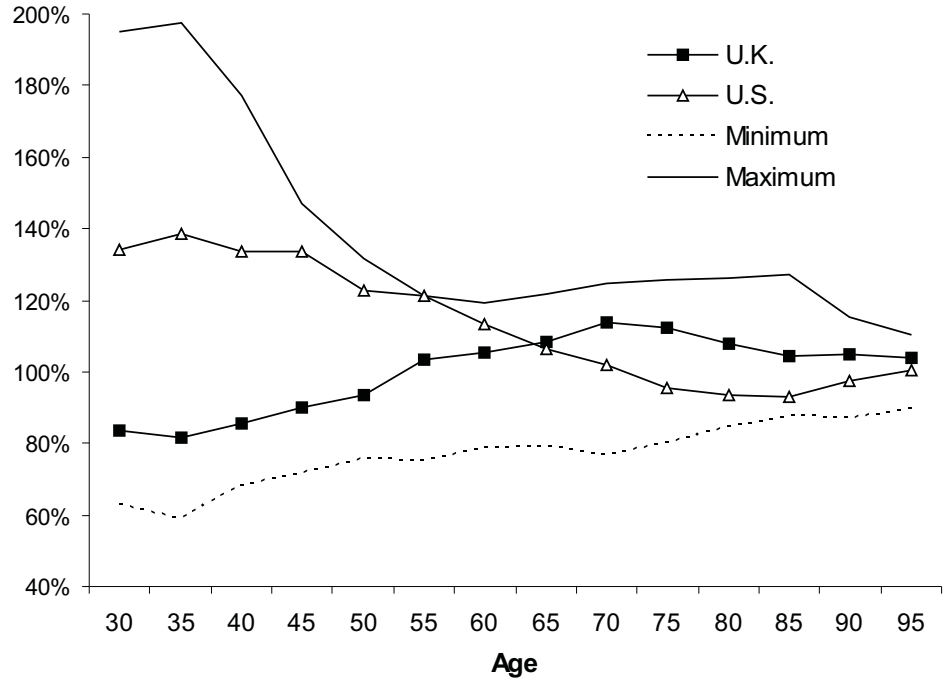

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Figure 3.2b. Male mortality rates by age relative to average. The 'average' international table is as per Figure 3.2a

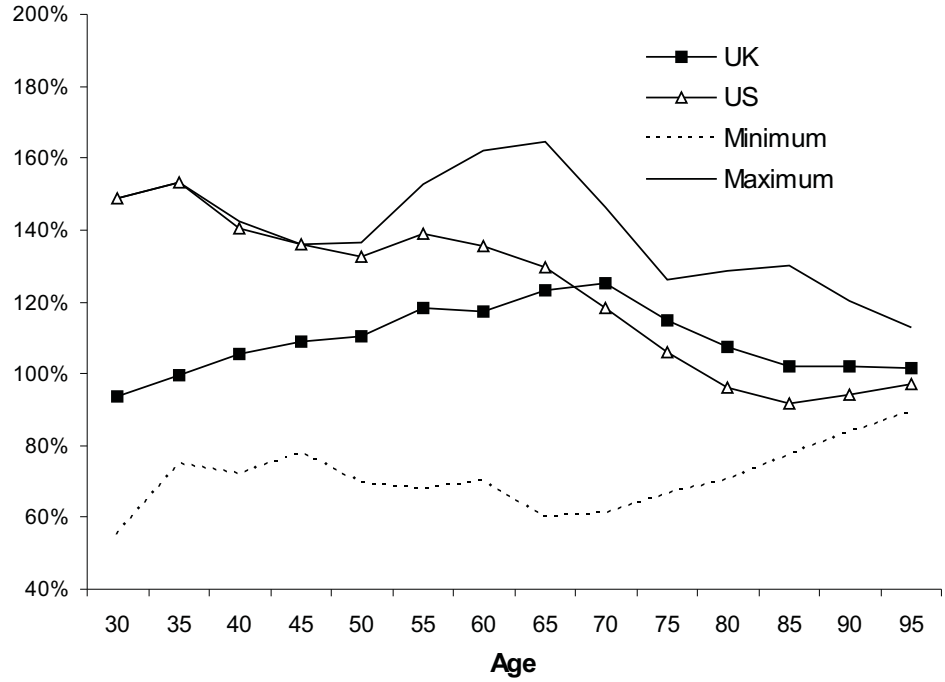

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Figure 3.2c. Female mortality rates by age relative to average. The 'average' international table is as per Figure 3.2a

Table 3.2. Ratio of mortality rates in the U.K. to those of the country with the lowest rates at each age

	Age				
	60	65	70	75	80
Male	134%	137%	148%	140%	128%
Female	168%	205%	205%	172%	152%

Own figures — data source: W.H.O.

age (Japan). The equivalent ratios for females are even higher. The U.K rate is more than double the rate for Japan for women aged 65 and 70.

3.3 *Life Expectancy at Age 65*

3.3.1 The U.K.'s relatively high mortality rates for elderly lives can also be illustrated by looking at how life expectancies at age 65 vary by country. Table 3.3 shows that life expectancies for people aged 65 in the U.K. are low relative to most comparable countries. It should be noted that the life expectancy figures are 'period' life expectancies, based on observed mortality

Table 3.3. Expectation of life (years) at age 65 for selected countries in 2000

Country	Males	Females
Japan	17.50	22.40
France	17.19	21.63
Switzerland	16.77	20.93
Australia	16.73	20.23
Sweden	16.65	20.01
Israel	16.64	18.87
New Zealand	16.56	19.93
Italy	16.46	20.57
Spain	16.22	20.23
United States of America	16.02	19.15
Canada	15.95	19.75
Singapore	15.92	18.65
Greece	15.91	18.56
Norway	15.79	19.68
Belgium	15.70	19.65
Austria	15.66	19.61
Denmark	15.27	17.77
Netherlands	15.13	19.54
Finland	15.07	19.18
United Kingdom	*15.06*	*18.54*
Germany	15.06	18.91
Portugal	14.31	18.01
Ireland	14.25	18.05

Source: W.H.O.

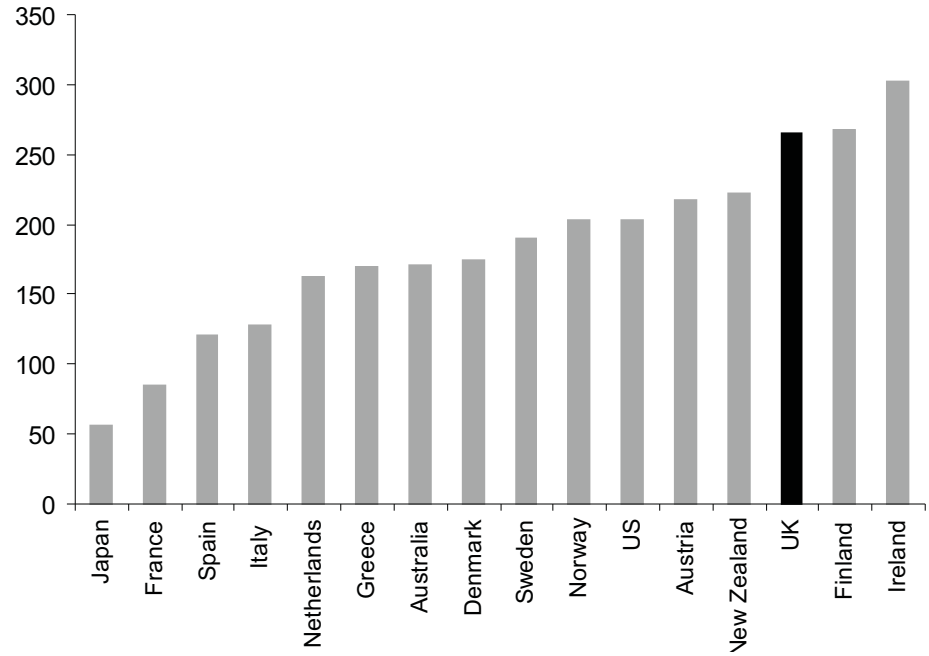
rates in the year 2000. They are therefore consistent with the values shown in Tables 2.2c and 2.2d in making no allowance for future improvements in mortality.

3.3.2 The difference between the U.K. and France is particularly stark. Men aged 65 in France can expect to live two years longer than men of the same age in the U.K. For women the difference is over three years, with French women having a 17% greater expectation of life at age 65.

3.4 *International Variations in Heart Disease Mortality*

3.4.1 The main reason for the U.K.'s relatively poor performance at older ages is that death rates from circulatory disorders, especially heart disease, are particularly high in the U.K. Age-standardised mortality rates for this cause for men aged 35-74 from a range of selected countries are shown in Figure 3.4.

3.4.2 An example of the U.K.'s relatively high rates can again be seen through a comparison with France. Figure 3.4 shows that the age-standardised rate of heart disease mortality for men in the U.K. was 312% of the equivalent rate in France. For women in the same range, the equivalent figure was 424%. (British Heart Foundation, 2003)



Data source: British Heart Foundation (2003)

Figure 3.4. Age-standardised death rates per 100,000 from coronary heart disease in 1998, selected countries, males aged 35-74

3.5 *International Variations in Cancer Mortality*

3.5.1 Overall cancer mortality rates in England and Wales are close to the Western European average (Department of Health, 1999). However, death rates from cancers affecting females, notably breast cancer and cervical cancer, are amongst the worst in Western Europe. This is the case despite the improvements discussed in Section 2.

3.5.2 Cancer survival rates are also relatively poor for many of the main causes of cancer. Five-year survival rates for lung, breast and colon cancer are all significantly worse than equivalent European Union average and U.S. figures. In an article in the *British Medical Journal* in 1999, Karol Sikora used the following description to illustrate the extent of the problem (Sikora, 1999):

> "If Britain could achieve the survival rates of the best country in Europe for each cancer over 25,000 lives a year would be saved. Even if it could just reach the European average, nearly 10,000 lives would be saved."

3.5.3 Recently published figures (Eurocare, 2003) also show that the U.K. lags behind other countries in Europe in terms of cancer survival rates. Figures for sample countries for various cancer sites are given in Table 3.5. It should be noted that figures of this kind should be interpreted with care as survival statistics can also reflect differences in screening and detection.

3.6 *Implications*

3.6.1 Mortality rates vary by country for a number of reasons: genetic variation, differences in diet and varying standards of healthcare are obvious examples. The extent to which rates in different countries will converge is open to debate. However, the fact that the U.K. has high relative mortality rates for elderly lives, tells us one important thing: U.K. experience has the clear potential for further significant improvements.

3.6.2 It is impossible to argue that we are close to hitting some kind of

Table 3.5. Five-year cancer survival probabilities for selected European countries

	Percentage of patients alive five years after diagnosis — by cancer site and gender			
	Prostate	Breast	All (F)	All (M)
England	54%	74%	51%	37%
Scotland	54%	72%	50%	36%
Wales	49%	70%	47%	35%
France	75%	81%	58%	45%
Germany	76%	75%	56%	44%
All Europe	65%	76%	54%	41%

Data source: Eurocare (2003)

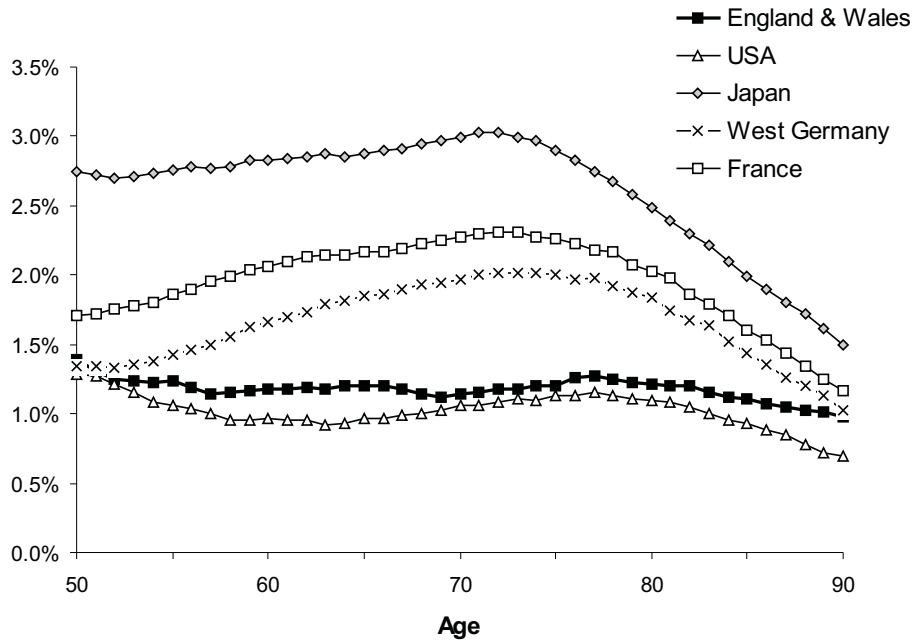
biological barrier that is going to prevent future improvements. Moreover, the potential for further improvements is greatest for ages between 60 and 80. It may not be a coincidence that a projection of the cohort trend that we have observed would lead to substantial improvements at just these ages in the first two decades of the 21st century.

3.7 *Variations in Rates of Mortality Improvement by Country*

3.7.1 The Mortality data used in this section were taken from the Human Mortality Database (www.mortality.org) maintained by the University of California, Berkeley (U.S.A.) and the Max Planck Institute for Demographic Research (Germany).

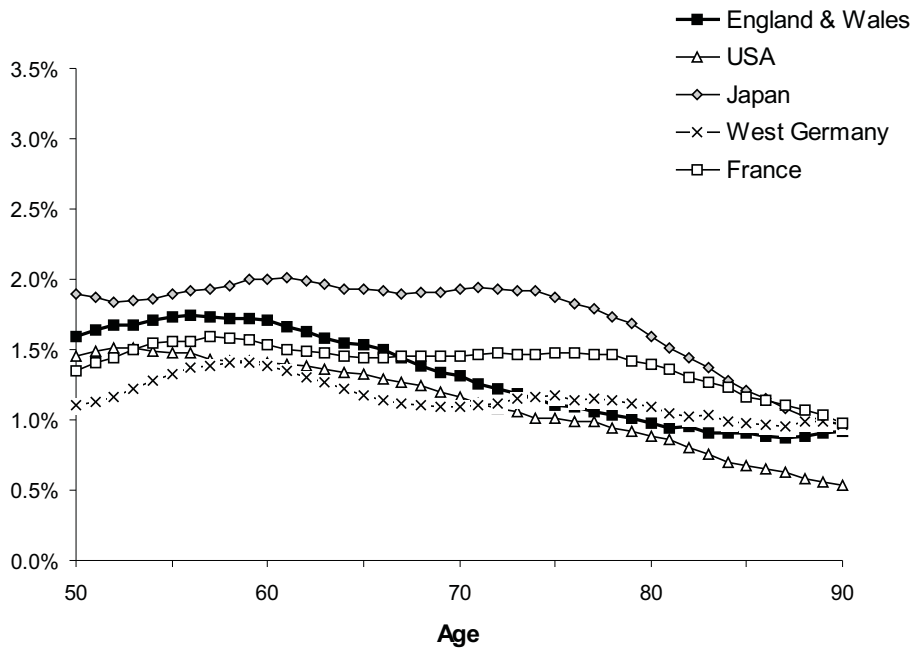
3.7.2 Figures 3.7a and 3.7b show how mortality improvements for the population of England and Wales have compared with some sample countries during a 40-year period from the late 1950s to the late 1990s. The actual periods, using the most up to date tables available on the Human Mortality Database, were to 1998 for England and Wales, 1999 for U.S.A., Japan and West Germany and 1997 for France.

3.7.3 Figure 3.7a shows that rates of improvement for females in England and Wales have been slightly higher, at all ages, than in the U.S.A. over the same period. However, mortality rates in France, West Germany,



Data source: www.mortality.org (2003)

Figure 3.7a. Average annual rates of mortality improvement for females over a 40-year period ending in the late 1990s by age and country, smoothed by age using seven-year rolling averages



Data source: www.mortality.org (2003)

Figure 3.7b. Average annual rates of mortality improvement for males over a 40-year period ending in the late 1990s by age and country, smoothed by age using seven-year rolling averages

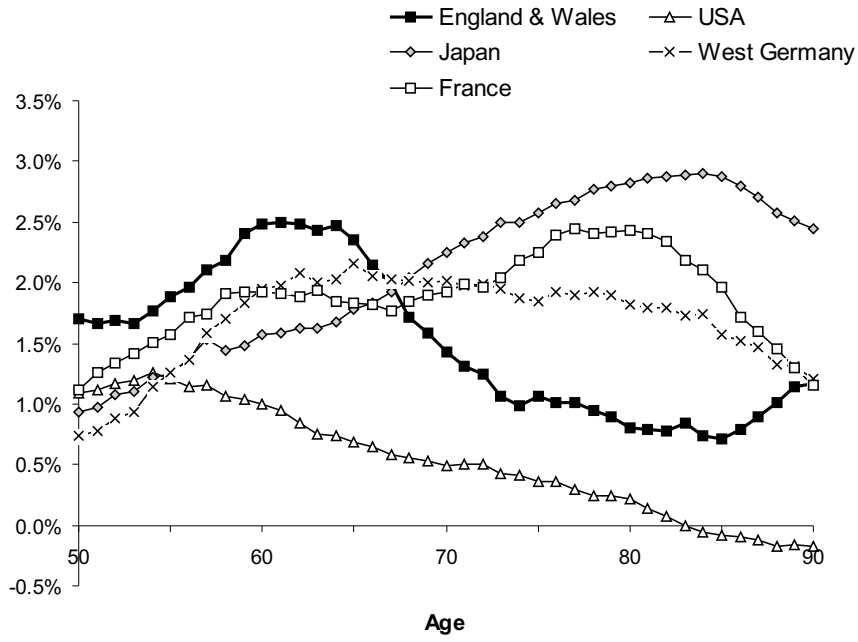
and especially Japan, have improved far more rapidly. The difference has been especially marked between ages 60 and 80. In the case of Japan the average improvement rate has been more than twice the equivalent rate for England and Wales for all ages between 50 and 80.

3.7.4 Figure 3.7b shows that rates of improvement for males in England and Wales have also been lower, at all ages, than in Japan. However, relative to the other countries, improvements have been high at ages up to 65, but relatively low at ages in excess of 75.

3.7.5 Figures 3.7c and 3.7d chart rates of improvement by age for the same countries over the latest ten-year, rather than 40-year, period. A number of features are apparent from these graphs of more recent mortality change. Firstly, the improvements for England and Wales are amongst the highest for ages 50 to 70 for both males and females. This is consistent with the observation of the cohort effect in the U.K. — i.e. the wave of rapid improvements affecting people born in the period 1925-45.

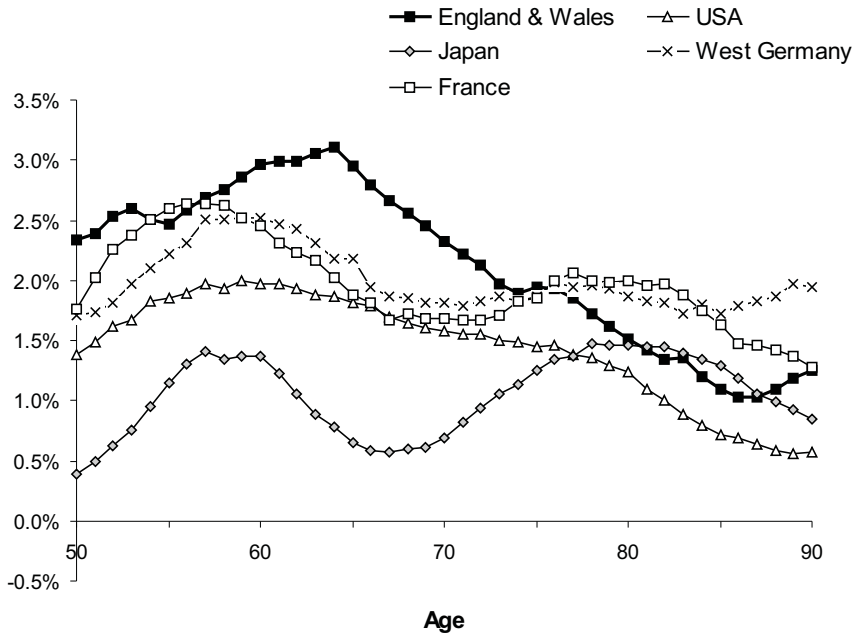
3.7.6 At ages above 70 the improvements for England and Wales have been around the average for males and at the lower end for females.

3.7.7 The experience for Japan over the recent period does not stand out nearly so much. In fact, for Japanese males the improvements are the lowest for the countries considered for ages 50 to 80. Improvements for Japanese females are still at the upper end, particularly above age 75.



Data source: www.mortality.org (2003)

Figure 3.7c. Average annual rates of mortality improvement for females over a ten-year period ending in the late 1990s by age and country, smoothed by age using nine-year rolling averages



Data source: www.mortality.org (2003)

Figure 3.7d. Average annual rates of mortality improvement for males over a ten-year period ending in the late 1990s by age and country, smoothed by age using nine-year rolling averages

3.7.8 Improvements for the U.S.A. are at the lower end of the spectrum and it is particularly noticeable that for females aged above 80 mortality rates have been largely static over the ten-year period. For females over age 85, mortality rates actually increased over the period in question.

3.8 *International Evidence of Cohort Effects*

3.8.1 There are a number of examples of birth cohorts in countries other than the U.K. that have experienced more rapid mortality improvement (MacMinn, 2003).

3.8.2 Willets (2003) discusses the example of trends in Japanese mortality experience in detail. The Japanese example is interesting, because there is evidence for a cohort of lives born around 1915, which has experienced more rapid improvement than earlier or later generations.

3.8.3 Willets took Japanese data from the Human Mortality Database (www.mortality.org). The original data were supplied by the Japanese Ministry of Health and Welfare and Japanese Statistics Bureau. Log linear regression was used to derive average rates of mortality improvement for successive nine-year periods for all ages between 50 and 95. For example, for age 80 the improvement rate for year 1995 was calculated by fitting a straight line to log mortality rates for 80 year olds for years 1991 to 1999. This methodology was used to produce a table of improvement rates for nine-year periods centred on years from 1970 to 1995. For each calendar year, the maximum rate of improvement (for any age) was derived. Table 3.8 shows only those improvement rates in excess of 95% of the maximum for each calendar year. The clear diagonal pattern shows that the highest improvements in each calendar year have been experienced by females born in the same period (roughly 1910-1925).

3.8.4 This is notable because this generation has continued to experience rapid improvement far into old age. This does not prove that the U.K. cohort effect will also continue to advanced ages. However, it does indicate that this is possible and it does counter arguments that year of birth effects will inevitably wear off with age.

3.9 *International Evidence of the 'Ageing of Mortality Improvement'*

3.9.1 In Section 2.18 — which discussed the 'ageing of mortality improvement' — it was noted that accelerating rates of mortality improvement have been observed at older ages in a number of countries around the world.

3.9.2 Kannisto (1994) charted the progress of mortality improvement in those aged between 80 and 99 in nine countries over the period 1950 to 1990 (the countries were Austria, Belgium, England and Wales, France, West Germany, Japan, Scotland, Sweden and Switzerland). He found that mortality improvements at these advanced ages were relatively low in the 1950s and 1960s, averaging around 0.5% p.a. to 1.0% p.a. However, since

Table 3.8. Average rate of mortality improvement (percent per annum) over successive periods of nine years centred on years 1970 to 1995, Japanese females aged 50 to 95, only values in excess of 95% of the maximum for each calendar year are shown

	Year																									
Age	'70	'71	'72	'73	'74	'75	'76	'77	'78	'79	'80	'81	'82	'83	'84	'85	'86	'87	'88	'89	'90	'91	'92	'93	'94	'95
50																										
51																										
52																										
53								4.6																		
54	3.7																									
55																										
56																										
57																										
58	3.8	4.1	4.5	4.6			4.5																			
59	3.7																									
60		4.0																								
61		4.0	4.3	4.6	4.8																					
62				4.5	4.6																					
63			4.5	4.6	4.9	4.8	4.5																			
64				4.4	4.7	5.0	4.7	4.5																		
65							4.7	4.5																		
66								4.4																		
67								4.5	4.9	4.5																
68								4.4		4.3																
69											4.1															
70										4.4	4.3	4.3														
71													4.5	4.7												
72												4.4	4.6	4.5												
73															4.8	4.5										
74														4.5		4.4										
75																4.4	4.2									
76																4.3	4.1	4.1	3.5							
77																			3.4							
78																			3.5	3.3	3.1					
79																			3.4		3.2					
80																					3.2	3.0				
81																					3.1	3.1	3.7			
82																					3.1	3.1	3.7	3.7		
83																							3.6	3.7	3.9	
84																								3.6	4.0	3.7
85																									4.1	3.7
86																										3.7
87																										
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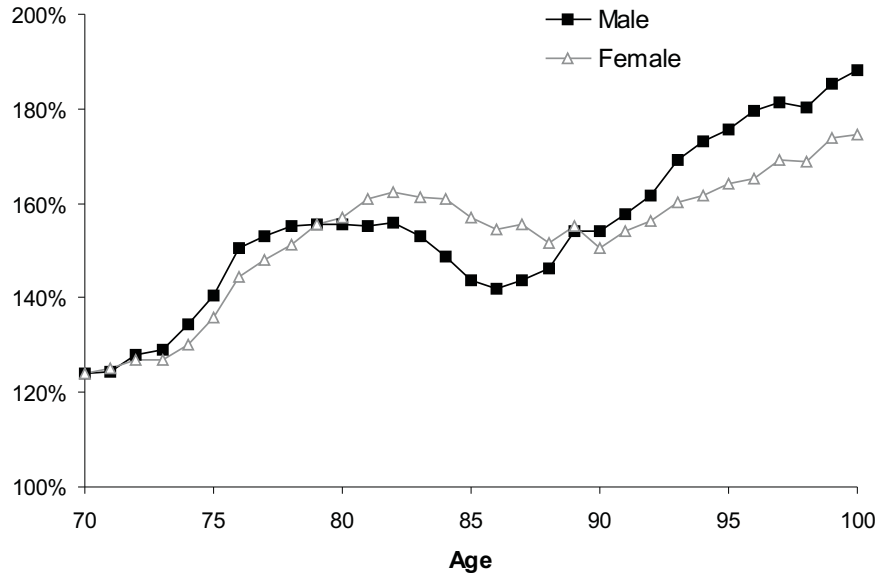
95																										

Data source: www.mortality.org

then, rates of improvement have been steadily accelerating. For instance, in the period 1980-89 the average rate for women aged 80 to 89 was in excess of 2.0% p.a.

3.9.3 In another study Wilmoth & Lundström (1996) analysed trends in the maximum reported age at death in Sweden, a country which has had reliable mortality data for a long period of time. They found that the maximum age has risen steadily for at least the past 130 years. This has — of course — been largely due to the increasing number of elderly people in the Swedish population. However, the increasing trend is showing no signs of deceleration.

3.9.4 The same data used to produce Figures 3.7a to 3.7d were also used to derive the values shown in Figure 3.9. Specifically, for the five sample countries (i.e. England and Wales, U.S.A., Japan, France and West Germany), average rates of improvement were calculated for the last ten years (of available data) and the previous 30 years, and ratios of the two values were derived. Figure 3.9 shows that for ages above 70 all of these ratios are in excess of 120%. For ages above 75 most of the ratios are close to, or above, 150%. It is notable that the highest values are for the oldest males. This further demonstrates the fact that mortality is improving at an accelerating rate at older ages.



Data source: www.mortality.org (2003)

Figure 3.9. Ratio of average annual rate of mortality improvement over last 10 years versus rate from previous 30 years. Average value for five sample countries (England and Wales, U.S.A., Japan, France and West Germany)

3.10 *A Maximum Human Lifespan — the International Debate*

3.10.1 The amazingly rapid pace of mortality improvement seen in Japan and the observation of accelerating improvements at older ages, suggest that a maximum theoretical human life expectancy (if such a thing exists) is not immediately in sight.

3.10.2 In the early 1980s — when Bernard Benjamin presented his paper 'The Span of Life' (1982) to the Institute of Actuaries — the arguments in favour of limits and limited improvements in elderly mortality seemed to be in the ascendancy. Benjamin's own view seemed more open-minded. He suggested that:

> "... the possibility exists that either genetically or by dietary intervention some extension may occur in the allegedly maximum lifespan of the human species."

Benjamin's paper, and the record of the subsequent debate at the Institute, revealed a substantial diversity of opinion on the issue.

3.10.3 Today the argument still rages amongst demographers on the international stage. Those in the 'optimistic' camp suggest that there is no foreseeable limit to life expectancy and tend to focus on the empirical evidence of accelerating rates of improvement.

3.10.4 In 1996 a project was initiated by the Society of Actuaries in the U.S.A. to assess the impact of mortality improvement on social security in Canada, Mexico and the U.S.A. (Sze & Rosenberg, 1998). The first phase of the project was a review of existing work and literature in the field of longevity and mortality improvement. A very comprehensive review was produced by Tuljapurkar & Boe (1998). In their report they argue that there is little basis, in theory or observation, for the existence of an irreducible component of mortality and substantial evidence against the existence of a precisely defined limit.

3.10.5 In a paper published in *Science* 'Broken limits to life expectancy' (Oeppen & Vaupel, 2002) historical predictions of limits to life expectancy are analysed. The limits set by demographers have consistently proved to be wrong and have tended to be broken within a short space of time. Instead, the highest observed life expectancy of any country has increased at a remarkably constant and linear rate.

3.10.6 The opposing view is that there are inherent biological limitations to life expectancy. A few decades ago this theory was the dominant one. It is less universally held now, but still has many advocates. Proponents of this view — such as Hayflick and Olshansky — still believe that substantial reductions in mortality are possible. However, they argue that these will come from the elimination of deaths from age-related diseases such as heart disease and cancer. Life expectancy will increase, but the potential increase will be limited by inevitable processes of ageing and damage accumulation.

3.10.7 In Sections 2.5 and 2.6 the variation of cause of death by age in

England and Wales was examined. Tables 2.5b and 2.6b showed that, for people aged *80 and over*, cancer and circulatory disorders (such as heart disease and stroke) account for 65% of deaths for men and 59% for women. Senility without psychosis (or 'old age') was recorded as a cause of death for just 2% and 6% of men and women respectively.

3.10.8 Medical developments and changes in behaviour (such as reduced smoking and better diet) are likely to lead to significantly lower rates of mortality amongst the elderly. However, assessing the precise impact of improvements in particular causes is a complex task. If circulatory disorders and cancer were eliminated as possible causes of death then mortality rates for men aged 80 plus would not reduce by 65%. Studies which attempt to show the numbers of years of extra lifespan produced by the elimination of various causes of death can be very misleading.

3.10.9 With increasing age, mortality rates from different causes become increasingly correlated with each other. More frail individuals become vulnerable to a whole host of medical conditions. If particular causes of death are eradicated, elderly people may well succumb to other causes. Put another way, reduced death rates from one cause may cause increased rates from other causes.

3.10.10 It is clear that substantial improvements in life expectancy at retirement age are possible through the elimination of the major causes of death at advanced ages, such as heart disease. However, potential lifespans of 130 years or more would clearly only be possible if the ageing process itself were understood and could be reversed. The current state of anti-ageing research is discussed in Section 4.

4. Medical Advances

4.1 *Introduction*

4.1.1 In Section 2 it was observed that a substantial part of current mortality improvement is being driven by advances in medicine. The reduced number of heart disease deaths has been partly due to the development of new treatments, such as beta-blockers, and new surgical procedures, such as bypass grafts and angioplasties. Improvements in cancer mortality have been largely due to advances in detection and treatment of cancers: underlying incidence rates appear to have remained broadly level or increased for many cancer types.

4.1.2 At the beginning of the 21st century, the results of scientific development are increasingly altering the way we live our lives. A prime example was the project to decode the entire human genome, which has provided us with a map of the DNA making up our chromosomes. This task was only possible because of the enormous developments in computing technology that have occurred over the past few decades. The human genome

project is now likely to sow the seeds for a whole range of scientific and medical progress.

4.1.3 The battle against cancer is progressing on a number of fronts, with much of the research having a genetic basis (one aspect of this is discussed later in this section). The growth of replacement organs for transplantation is another area in which progress is likely in the 21st century and new surgical procedures for combating heart disease are also likely.

4.1.4 The pace of scientific development appears to be accelerating and it is possible that this explosion in knowledge will drive increasingly rapid advances in medicine. These advances may cause mortality rates to fall with increasing speed.

4.1.5 A comprehensive analysis of future trends in medicine is beyond the scope of this paper. However, two particular areas of medicine will be explored. Firstly, a potential development in the treatment of cardiovascular disease and, secondly, research into the ageing process.

4.2 *Drug Treatments for Cardiovascular Disease*

4.2.1 In this section some recent developments in the treatment and prevention of heart disease and stroke are discussed. These developments are of considerable interest in themselves and also provide some insight into how medical advances may occur in future. Heart disease and stroke are major causes of mortality in the U.K. In Sections 2.5 and 2.6 we saw that over 40% of deaths for people aged over 70 in England and Wales are due to circulatory disorders, such as heart disease and stroke. Clearly, any developments which reduce the incidence of heart disease and stroke are likely to have a major impact on population mortality.

4.2.3 Some of the developments discussed in this section have been established as medical practice for a number of years. However, the most recent development has not yet even been tested. This was announced in a series of three papers in a June 2003 issue of the *British Medical Journal*, one of which was entitled, 'A strategy to reduce cardiovascular disease by more than 80%' (Wald & Law, 2003). These papers were highlighted by two editorial articles in the same issue, entitled, 'A cure for cardiovascular disease?' (Rodgers, 2003), and, 'The most important BMJ for 50 years?' (Smith, 2003). These dramatic announcements were, understandably, picked up by the popular press; the *Times* on 27 June 2003 had an article with the headline '£1 'superpill' could cut heart attacks by 80%' (Hawkes, 2003).

4.2.4 The major risk factors for heart disease and stroke are well known: age, sex, smoking, obesity, lack of exercise, high blood sugar level (diabetes), high blood pressure and high cholesterol level. Extensive public health campaigns have been aimed at getting each of us to reduce our risk of heart disease and stroke by giving up smoking, taking more exercise and improving our diet *whether or not we are in a high risk category*. The strategy for addressing increased risk due to diabetes, high blood pressure and high

levels of cholesterol is different. In these cases, a 'threshold' strategy has been adopted. At the risk of oversimplification, this means that treatment is recommended for an individual when, for example, their blood sugar level exceeds a given threshold. The precise level of this threshold will depend on general medical considerations, the particular medical profile of the individual and the cost of the treatment, among other factors.

4.2.5 High blood pressure has long been recognised as a risk factor for heart disease and stroke and effective therapies have been available since the 1950s (Brackenridge & Elder, 1998). According to Law *et al.* (2003):

> "lowering systolic blood pressure by 10mm Hg or diastolic blood pressure by 5mm Hg reduces the risk of stroke by about 35% and that of ischaemic heart disease ... events by about 25% at age 65."

In their paper, these authors carry out a meta-analysis of 354 trials involving the five main types of blood-pressure-reducing drugs: thiazides, beta blockers, angiotensin converting enzyme (ACE) inhibitors, angiotensin II receptor antagonists and calcium channel blockers. They conclude that:

> "All five categories of drugs produced similar reductions in blood pressure. The average reduction was 9.1mm Hg systolic and 5.5 mm Hg diastolic at standard dose ..."

4.2.6 Statins are a relatively recently developed class of drugs, and were first marketed in the late 1980s. They are designed to lower levels of cholesterol in the blood. Cholesterol consists of high density lipoprotein (H.D.L.) and low density lipoprotein (L.D.L.) together with triglycerides, see Brackenridge & Elder (1998). High levels of H.D.L. and low levels of L.D.L. are beneficial. Conversely, low levels of H.D.L. and high levels of L.D.L. are harmful. Statins act specifically to reduce L.D.L. Brackenridge & Elder (1998) state that:

> "Dr William Roberts, editor of the American Journal of Cardiology, has compared the statin class of drugs with antibiotics in terms of the influence they will have on modern medicine."

4.2.7 High levels of cholesterol, particularly L.D.L., are a well-established risk factor for heart disease. Their role as a risk factor for stroke is less certain. Law, Wald & Rudnicka (2003) state that:

> "... cohort studies showed no association between serum cholesterol concentrations and stroke."

This is consistent with the findings of Macdonald *et al.* (2003) based on data from the Framingham Heart Study. However, Law, Wald & Rudnicka (2003) also report what they refer to as a 'paradox', in that earlier studies "... showed that statins reduced the incidence of stroke by about 30%."

4.2.8 The claims that cardiovascular disease could be reduced by more than 80%, mentioned above, are based on the research of Professors Law and Wald. Their concept, for which they are currently seeking a patent, is appealingly simple. They propose that a single pill, consisting of six drugs already used individually to treat risk factors for cardiovascular disease, should be taken by everyone over the age of 55 irrespective of their pre-treatment levels of these risk factors. The ingredients of this 'Polypill', a name for which Professors Law and Wald have applied for a trademark, are:

— a statin to reduce L.D.L.;
— a combination of low doses of three blood pressure reducing drugs;
— folic acid to reduce the level of homocysteine in the blood; and
— aspirin to regulate blood platelet function.

High levels of homocysteine and irregular blood platelet function are risk factors for cardiovascular disease (Brackenridge & Elder, 1998; Wald & Law, 2003). It is interesting to note that ACE inhibitors (anti-hypertensive drugs that block the formation of angiotensin in the kidney, leading to relaxation of the arteries) are also useful in the treatment of kidney disease (see, for example, Terajima *et al*. (2003)).

4.2.9 Wald and Law's research involved the meta-analysis of 354 trials of five types of blood pressure reducing drugs referred to above and three meta-analyses involving a total of 280 trials of statins (see Law *et al*., 2003; Law, Wald & Rudnicka, 2003). Points put forward by Wald & Law to support their case include:

— The effect of combinations of drugs to reduce blood pressure was additive. This was "not surprising as the different categories of drugs have different modes of action, apart from ACE inhibitors and angiotensin II receptor antagonists." (Law *et al*., 2003).
— All five drugs to reduce blood pressure had similar effects; an average reduction in systolic blood pressure of 4.4 mm Hg at half standard dose. (Law *et al*., 2003).
— "The prevalence of ... [side effects] with two [blood pressure reducing] drugs in combination was less than additive" (Law *et al*., 2003).
— "Statins significantly lowered L.D.L. cholesterol from all pre-treatment concentrations" (Law, Wald & Rudnicka, 2003).
— The "combined effect of changing the four risk factors (the effect of the Polypill) [was calculated] by multiplying the relative risks associated with each" (Wald & Law, 2003).

4.2.10 One feature of this proposal likely to be controversial is that it involves recommending drugs to everyone over the age of 55 regardless of their state of health. In other words it is not based on a threshold strategy, unless age is regarded as a risk factor and 55 is then taken to be the threshold. Other features worth noting are:

— "No trial has studied the effect of three [blood pressure reducing] drugs in combination" (Law *et al.*, 2003). The assumed additive effect of combining three different blood pressure reducing drugs is an extrapolation from trials involving two drugs.
— The effects of the four different types of drug are assumed to be independent. This is the final point noted in Section 4.2.9. This assumption has not yet been tested.

Rodgers (2003), commenting on this exciting proposal, says, 'cost will be the key.' Important factors here are that the constituent drugs for the Polypill are all either off-patent or soon will be. This will make the Polypill cheap for any pharmaceutical company to produce. However, as Smith (2003) says: " ... large pharmaceutical companies are not keen. Not only will their profit margin be low but many of their expensive drugs may be made redundant."

4.2.11 The future effects on mortality and morbidity from the use of statins will be of considerable interest to actuaries. The effect of the Polypill, if it meets Professors Law and Wald's expectations, will be of even greater interest. It should be noted that many in the medical community have remained somewhat sceptical (White, 2003). However, if nothing else, the work of Law and Wald offers us a potential insight into how preventative medicine may lead to reduced mortality in the future.

4.3 *Ageing*

4.3.1 In this section we briefly discuss research into human ageing. This is a huge topic and has been the subject of many papers and books published in recent years. For instance, a paper by Gene Held (2002) provides a more comprehensive review. The ageing process works on a number of levels. We know *what happens* to our bodies as we age and we believe we know *why* we age. However, we do not know precisely how the ageing mechanisms within our bodies work. To quote Michael Horan (1998), Professor of Geriatric Medicine at the University of Manchester:

> "A precise explanation of what causes the ageing of an individual ... and the processes that bring it about is not yet possible, though we have strong evidence that they are in part genetically determined."

4.3.2 The following list is a brief summary, based largely on Medina (1996), of what happens to us as we age:

— The rate at which we shed skin cells becomes greater than the rate at which they are replaced, resulting in loss of tissue and increased susceptibility to creasing. Our skin also loses its flexibility making it more likely to sag.
— The rate at which bone is created is eventually exceeded by the rate at which it is eroded, resulting in a reduction of bone mass and structural

weakness. The rate of bone loss begins slowly (typically starting around age 40) and then accelerates with age, with women eventually achieving a rate of loss twice that of men.

— Our muscular abilities decline with age primarily because we lose muscular mass. The death of muscle cells causes total strength to reduce by roughly 30% to 40% by the time we reach our 70s or 80s.

— Peak human flexibility usually occurs before age 20. This is followed by a steady decline in joint function and restriction of mobility.

— As we age the nerves in our brains die. We lose between 30,000 and 50,000 a day, so that between the ages of 20 and 65 almost 10% of our brain cells die, with no possibility of regeneration. Some functions of the brain are noticeably affected, for instance short-term memory often deteriorates with age. Other parts of the brain hardly change at all. An example is the thalamus, which is involved in the processing of sensory signals, which loses few cells. The effect of cell loss is highly debatable. The 'neural plasticity model' contends that the brain can compensate for lost cells, by establishing new connections between neurons.

— The effectiveness of the heart as a 'pumping machine' gradually reduces with age. The fastest heart rate attainable during exercise falls and aerobic power (the amount of oxygen we can take in during exercise) declines by about 30% to 40% in the years up to age 65. One major reason for this decline is that the walls around the left ventricle of the heart thicken with age.

— The ageing of our lungs is multi-factorial. Loss of 'elastic recoil', combined with problems with muscles, nerves and blood vessels, cause a 50% decline in the oxygen-to-tissue transfer rate by age 70.

— Relatively speaking, the stomach, liver, pancreas and colon are relatively unaffected by advancing age. On the other hand, the small intestine loses the ability to absorb certain nutrients. The kidneys lose mass and function, resulting in less effective removal of waste products from blood (by the time we reach age 90 almost half the capability is lost). And, finally, the bladder's capacity to store urine reduces and its function worsens.

— Through the years, more and more layers of tissue are added onto the outside of the eye's lens. As a result, by age 70, the mass of the lens has almost tripled and the diameter has increased accordingly. This causes us to become more far-sighted.

— Cell death — starting around puberty — eventually leads to the loss of the perception of specific frequencies of sound, with the higher tones first to go.

— In women, the menopause is heralded by complex hormonal interactions, usually beginning between the ages of 45 and 50, which cause the ovaries to stop producing oestrogen. The ability of men to reproduce declines more gradually with age.

This, rather depressing, list suggests that:
— ageing is a very complex, multi-factorial process; and
— the effects of ageing on the body are quite haphazard, with some functions severely affected and others hardly impaired.

4.4 *Why We Age*

4.4.1 Section 4.3 described *how* we age. Historically, there have been two sets of 'evolutionary' theories that have sought to explain *why* ageing occurs. The first describe the presence of 'ageing genes.' These are genes which are selected through evolution because they confer a direct advantage to the *species'* survival, but at the expense of individual death. In other words ageing is seen as a deliberate and active process. These theories are given little credence today (Kirkwood, 1998).

4.4.2 Most weight is now given to the second set, so-called 'non-adaptive' evolutionary theories. These theories do not suggest ageing confers any benefit *of itself*. Instead senescence is explained through the *indirect* action of natural selection. Three examples of non-adaptive theories are as follows:

(1) 'Mutation accumulation' theory, which stems from the observation that natural selection is relatively powerless to act on genes which express their effects late in the lifespan, after the period in which reproduction is most common. Thus, ageing is explained by the accumulation over a large number of generations of harmful, late-acting mutations.
(2) 'Antagonistic pleiotropy' theory, which holds that ageing results from trade-offs between genes which confer early-life fitness benefits at the expense of late-life fitness disadvantages.
(3) 'Disposable soma' theory, which has its basis in the allocation of metabolic resources between the activities of growth, somatic (i.e. bodily) maintenance and reproduction. Models have shown that the optimum allocation strategy results in smaller investment in bodily maintenance than would be required for indefinite lifespan.

The haphazard aspects of ageing seem consistent with the explanations provided by non-adaptive evolutionary theories. However, it is apparent that there is a huge theoretical gulf between these evolutionary theories of ageing and the description of what happens to our bodies as we age. The gap is an understanding of the exact mechanisms that are involved in the ageing process. For example, we know that brain cells die as we age, but do not really know precisely what causes them to die (and why certain parts of the brain should be subject to cell loss and others not).

4.5 *Theories of Ageing*

4.5.1 Whilst there is a lack of complete understanding, there has been a lot of progress in the field of ageing research. There are plenty of ideas and a

large range of different theories. In his review paper, Held (2002) cited the example of the Russian gerontologist Medvedev who had reportedly listed and categorised over 300 theories of ageing. Some of these ideas overlap and others appear quite independent. One challenge facing researchers is the development of a 'unified theory of ageing.' A few decades ago this appeared to be a long way off. However, in recent years scientists have been getting more optimistic, as the quotes listed below demonstrate.

> "With the knowledge that is accumulating now about the nutritional and neuroendocrine aspects of ageing, and if we develop ways to repair ageing tissues with the help of embryonic cells, we could add 30 years to human life in the next decade. And beyond that, as we learn to control the genes involved in ageing, the possibilities of lengthening life appear practically unlimited."
>
> William Regelson — Professor of Medicine at the Medical College of Virginia — quotation in Medina (1996)

> "I believe ... in 25 years time we could see the creation of the first products that can postpone human ageing significantly. This would be only the beginning of a long process of technological development in which human life span would be aggressively extended. The only practical limit to human life span is the limit of human technology."
>
> Michael Rose — University of California — quotation in Medina (1996)

> "The cure for ageing must now be taken seriously by responsible gerontologists, because it is no longer science fiction."
>
> Aubrey de Grey (2003) — Department of Genetics, University of Cambridge

4.5.2 The work of Dr Aubrey de Grey has become more widely known amongst actuaries in the U.K. through his participation in a recent seminar (October 2003) on mortality improvement jointly sponsored by the C.M.I. Bureau and the GAD. In his presentation at the seminar de Grey described how the 'war on ageing' could be only a decade away and discussed what actuaries should be doing in the run-up to a 'post-ageing world.' He explained how advances in medicine could lead to 'engineered negligible senescence' and described a set of milestones on the path to achieving this goal. De Grey's theories are founded on the belief that there are only seven mechanisms for accumulating damage to the human body. Furthermore, therapies for reversing or obviating all of these types of damage are clearly foreseeable. He felt that, given sufficient commitment and resources, the goal of 'engineered negligible senescence' could be achieved by the year 2025.

4.5.3 Obviously, not everyone agrees with these views; but should we completely ignore what these scientists are saying? The general consensus suggests that we are unlikely to see a cure for ageing in the next few decades. However, looking further into the future — say 30 or 40 years — it is very difficult to tell whether the optimism of some scientists will prove to be correct. With the accelerating pace of scientific development, a great deal can

change in 30 or 40 years. Yet, the youngest members of final salary schemes may well be alive 60 or 70 years from now, perhaps even longer. So what risks are employers running by having final salary schemes? We will return to this issue in Section 7, which considers the implications of mortality change. The remainder of this section outlines some specific examples of ageing research, which have been discussed in recent years. This list is not exhaustive, but will give the reader a flavour for the kind of research that is taking place.

4.6 *Cell Division and Telomerase*

4.6.1 For decades, proponents of a maximum human lifespan have talked about the 'Hayflick limit.' Certain cells in the bodies of mammals constantly replicate by splitting in two, including those found in the stomach and skin. In the 1960s Hayflick found that mammalian cells could only divide up to a specific, limited number of times. The limits correlated roughly with the typical life spans of the organisms from which the cells came (Hayflick & Moorhead, 1961).

4.6.2 Recently it has been found that, in certain circumstances, the Hayflick limit can be overriden by the enzyme telomerase, which has been dubbed the 'immortality enzyme.' Telomerase has this effect because the 46 chromosomes found in each cell nucleus have protective ends called telomeres. Chromosomes are long strings of DNA containing the genetic blueprint of an individual. The telomeres are repetitive sequences of DNA which shorten each time a cell divides — they are sometimes described as being like the 'plastic tips of shoelaces, which eventually fray with age.' When the telomere becomes too short to protect the chromosome, the cell can no longer divide and eventually dies.

4.6.3 Certain cells in the human body are 'immortal' in the sense that they can divide indefinitely. Most cancer cells have this property, which is believed to stem from the presence of telomerase.

4.6.4 In 1998 it was found that adding telomerase to normal cells in culture allowed the telomeres to 'grow back,' greatly extending the lifespan of the cells (Bodnar *et al.*, 1998). The research team used human skin cells, cells from the retina of the eye and cells from inside arteries. All were able to grow back their telomeres.

4.6.5 It was feared that use of the telomerase enzyme might cause cells to become cancerous. This fear was dispelled by further research published in 1999 (Morales *et al.*, 1999).

4.6.6 Telomerase also played a part in two big science stories at the end of the 20th century. First, in May 1999, it was found that cells taken from Dolly the sheep (the first cloned mammal) had telomeres 20% shorter than cells taken from uncloned sheep of the same age (Shiels *et al.*, 1999). The DNA used to create Dolly was taken from a 6-year-old sheep. It appears that the shortened telomeres of the parent were inherited by the cloned sheep

and there was much speculation that Dolly would age prematurely. In February 2003 Dolly died, aged 6, having suffered from a 'progressive lung disease' and arthritis (BBC News Online, 2003a). Sheep can live for 11-12 years.

4.6.7 Second, in July 1999, a team of scientists created a human tumour in a culture dish for the first time. Only three genetic changes were required to turn a normal cell into a cancerous one. One of these steps was the switching on of the telomerase gene (Hahn *et al.*, 1999).

4.6.8 Some scientists feel that the potential of telomerase to increase human longevity has been exaggerated. Critics point to the fact that cell longevity has only been influenced *in vitro* and not *in vivo* (i.e. in a culture dish rather than in the body). It has also been suggested that telomere shortening may be more of a symptom of ageing than a cause (Rubin, 1998).

4.6.9 Telomerase may prove most useful in the treatment of cancer. For example, research by Szutorisz *et al.* (2003) showed that the 'telomerase gene' (hTERT) exists in a more accessible form in cancer cells than normal cells. The increasing understanding of how tumours activate telomerase may help in the development of drugs that target this process to restore mortality to cancer cells. BBC News Online (2003b) reported the establishment of a Europe-wide collaboration between research institutes and pharmaceutical companies to design drugs that will exploit the latest research and stop telomerase from being activated in cancer cells.

4.6.10 The potential of telomerase in the treatment of liver disease has also been discussed (Rudolph *et al.*, 2000).

4.7 *Longevity Genes*

4.7.1 The possibility of increasing longevity by genetic manipulation has been widely discussed in mainstream works of popular science as well as academic journals. For instance, Kaku (1998) suggests that in the next century:

> "... scientists will be searching for 'age genes' which may retard or repair molecular damage due to the ageing ... Assuming that such genes exist and can be isolated, then perhaps through gene therapy the process of ageing may be arrested and one's maximum life span be extended."

4.7.2 Genes linked to longevity — so-called 'gerentogenes' — have been isolated and manipulated in experiments on certain varieties of flies and worms. For example, in the nemotode — a tiny worm — the gene *age-1* has been isolated. This gene can be manipulated to increase lifespan by 110% (Friedman & Johnson, 1988). Of course, this does not necessarily mean that the same kind of gene manipulation will be possible in humans. Projecting biological findings from tiny creatures to humans is always problematic.

4.7.3 Non-adaptive evolutionary theories suggest that there may be

hundreds or thousands of 'gerentogenes'. However, some researchers believe there may be only a handful involved, perhaps as few as ten (Held, 2002). To complicate matters, it should also be noted that just because genes are *linked* to longevity does not mean they *cause* ageing. Furthermore, even if genes responsible for ageing *were* isolated, the next problem would be to devise some form of 'gene therapy' that could alter the genetic structure of all the billions of cells in a human body. Cells have actually developed sophisticated protection mechanisms to prevent such manipulation, so such therapy could be very difficult in practice.

4.7.4 Ageing appears to be such a complex, multi-factorial process that the discovery of one simple gene that can control the human lifespan, seems a remote possibility. However, with the rapid advancements evident in genetics, likely to be further enhanced by the project to decode the human genome, it is difficult to imagine that progress will not be made in this area in the 21st century.

4.8 *Error Accumulation and Free Radicals*

4.8.1 The idea that ageing may be caused by a gradual accumulation of 'wear and tear' on the body has been with us for some time. Among the culprits for this damage accumulation are 'free radicals', highly reactive atoms or groups of atoms that have at least one unpaired electron, created as by-products of the burning of oxygen in the body. Free radicals may react with other molecules, resulting in cascades of reactions that damage cells and prevent them from performing their normal functions. The body has developed various defence mechanisms to fight this process. The bio-chemicals produced to control the effects of free radicals are collectively termed 'anti-oxidants'. The mechanisms for producing antioxidants appear to break down with age, but it is not clear precisely *why* this breakdown occurs.

4.8.2 Since anti-oxidants are linked with fighting the effects of ageing, it is not surprising that they have been promoted as anti-ageing 'treatments'. It has been suggested that large doses of vitamins A, C and E (which are antioxidants) could help to promote longevity and a wide range of anti-oxidant treatments are available over the internet.

4.8.3 However, many believe there is little scientific evidence to suggest that anti-oxidant supplements work in practice (Austad, 1997) and there have been claims that the wrong quantities of anti-oxidants may actually increase the body's oxidative stress (Held, 2002).

4.8.4 In 2002 a position statement was issued by 52 researchers in the field of ageing (Olshansky *et al.*, 2002). The statement sought to inform the public of the distinction between the 'pseudo-scientific anti-ageing industry' and the genuine science of ageing. It claimed that the products of anti-ageing medicine, including anti-oxidants, have no scientifically demonstrated efficacy and may actually be harmful.

4.9 *DHEA and Other Hormone Therapies*

4.9.1 A lot of attention has been focussed recently on trying to understand the role of hormones in the ageing process. Certain hormones appear to have a significant impact on health and ageing. For instance, post-menopausal women have levels of oestrogen which are far below those of younger women. Hormone Replacement Therapy (HRT) — in which levels of oestrogen are boosted — has been linked with reduced incidence of heart disease and prevention of osteoporosis. Some studies have claimed that mortality rates in women taking oestrogen are about 50% of those not doing so (Austad, 1997). More recent research has questioned these findings and a negative feature of HRT is the reported link with a higher incidence of breast cancer.

4.9.2 Another hormone which has caused a lot of interest recently is dehydroepiandrosterone (DHEA) (Larkin, 1998). It is found in the human body in large quantities, but its function is unknown. What *is* known is that levels of DHEA decline rapidly after age 30. Some studies have described dramatic results from treating elderly people with DHEA. There have been claims of reduced joint pain, enhanced quality of sleep, improved mobility and increased muscle mass.

4.9.3 In a study published in the journal *Science*, Roth *et al.* (2002) report that the length of a person's life is related to three bio-markers: their body temperature and levels of insulin and DHEA. On average people who have lower body temperature, lower levels of insulin and higher levels of DHEA tend to live longer.

4.9.4 BBC News Online (27th July 2001) described the arrival of DHEA anti-ageing medicine in France as follows:

> "In France ... the arrival of the DHEA pills was greeted by the nation with something resembling hysteria. It was one of the main cultural events of the year, provoking the sort of anticipation you would normally get at Britain's January Sales ... there were queues of people quickly forming outside chemist shops — eager for a chance to experience the elixir of youth."

4.9.5 Later research has poured cold water on the more extravagant claims (e.g. Flynn *et al.*, 1999) and it should be noted that *all* hormone levels actually decrease with age. However, it is not known whether reduced hormone levels are a cause or a symptom of ageing.

4.10 *Caloric Restriction*

4.10.1 One proven method of increasing longevity in laboratory animals is caloric restriction. A diet low in calories — i.e. just above starvation level — has been shown to increase the lifespan of mice and rats by between 50% and 100%.

4.10.2 It is not clear why this happens, or whether caloric restriction would have any beneficial effect on humans. One thing is certain. Few people are likely to attempt such a near-starvation diet in order to increase their

potential longevity. However, scientists are very interested to discover how caloric restriction works, in order to improve their understanding of the ageing process.

4.10.3 An ingredient of red wine, resveratrol, seems to mimic the age-enhancing effects of caloric restriction. It is one of a group of chemicals called polyphenols, which are claimed to protect against heart disease and osteoporosis. Howitz *et al.* (2003) found that the compound extended the lifespan of yeast by up to 70%. Resveratrol boosts levels of a protein called Sir2, which is thought to extend lifespan by stabilizing DNA.

4.10.4 Vellai *et al.* (2003) in the scientific journal *Nature* describe how eliminating an enzyme called TOR more than doubled the lifespan of nematode worms. TOR, which is also present in humans, is thought to regulate metabolism, sensing the availability of nutrients and translating that into protein synthesis and cell growth. The researchers suggest that caloric restriction results in low TOR expression and hence increased lifespan.

5. The Threat from Infectious Diseases

5.1 *Introduction*

5.1.1 This section of the paper summarises the main issues relating to the threat of infectious diseases. A more detailed analysis can be found in Appendix 2, where incidence and mortality rates from specific diseases are explored in depth.

5.1.2 Developments in science and technology have brought many benefits to society and some of these are discussed in Section 4. However, the other side of the coin is that many of these developments may also expose us to threats from infectious diseases.

5.2 *The Increased Threat from Infectious Diseases*

5.2.1 Rapid global transport, especially air travel, may allow a disease to be spread before it is recognised. This was clearly demonstrated by the spread of severe acute respiratory syndrome (SARS) to some 30 countries in less than four months in early 2003, killing 800 people in spite of unprecedented global co-operation. Cross-border travel and migration have also played a role in the spread of human immuno-deficiency virus (HIV) and tuberculosis (TB) within the U.K. population.

5.2.2 Extensive use of antibacterials in medicine and veterinary science has improved health care and farming productivity. However, it has also resulted in the emergence of disease-causing bacteria that are resistant to previously effective drugs.

5.2.3 Potential advances in xenotransplantation (transplant of animal tissues or organs to humans) may expose us to new diseases originating from animals (for a review see Muir & Griffin, 2001).

5.2.4 Changes in food production could expose the public to new diseases, as shown by the emergence of new variant Creutzfeldt-Jacob disease (vCJD) that has been linked to B.S.E. ('mad cow disease') in the U.K.. With increasing industrialisation of food production, problems with a single food producer or method of production may affect a large number of people.

5.2.5 Human behaviour has played a role in disseminating deadly infectious diseases. Intravenous injecting drug use has been a major route of transmission of the hepatitis C virus (HCV) in the U.K. and HIV in many parts of the world such as Eastern Europe. Unprotected sex and sexual relationships with multiple partners have contributed to the current HIV pandemic that has killed an estimated 3.1 million people worldwide in 2002 alone.

5.2.6 There is also now the possibility of bioterrorism, involving deliberate spread of fatal infectious agents such as anthrax and smallpox, which further highlights the potential threat of infectious diseases to the public (for a review see Beeching *et al*., 2002).

5.3 *Global Co-operation*

5.3.1 It is worth noting that advances in medicine, telecommunications and international networking will continue to help limit the effects of new infectious diseases. The experience from SARS has shown that effective international communication and efforts can result in rapid detection of new cases, hence preventing further infection. Measures set in place to prevent global influenza and other pandemics as well as bioterrorism will play important roles in containing outbreaks. Developments in antimicrobials, vaccines and detection methods will also be able to protect the public from the threat of infectious diseases.

5.3.2 Nevertheless, HIV remains a threat to global morbidity and mortality as well as to the global economy. In high-income nations, the use of highly active antiretroviral therapy (HAART) has postponed HIV-related death, but the number of newly infected individuals continues to rise. Furthermore, HAART has many side effects and the long-term effects on ageing HIV-infected individuals are still uncertain. The African continent is still the most affected region in the world, fighting the epidemic with limited resources. More recently, Eastern Europe and parts of Asia have become homes to some of the fastest growing HIV epidemics in the world.

5.4 *Conclusions*

5.4.1 Arguably, HIV is the only infectious agent to emerge in recent decades to have had a dramatic impact on global mortality. However, the emergence of antibacterial-resistant strains, vCJD, SARS and HCV shows that the threat posed by infectious diseases cannot be disregarded. Some

believe that we have been fortunate that a more devastating disease has not surfaced, such as one which can be spread as easily as the common cold but is as deadly as Ebola.

5.4.2 The latest C.M.I. Bureau term assurance experience shows rates of mortality for female non-smokers in their 30s of less than 1 in 3,000. Therefore, in relative terms, a small increase in infectious disease mortality could have a significant impact on aggregate mortality rates for younger adults. At older ages, infectious causes of death are likely to become more common (in relative terms) as deaths from cancer and heart disease reduce. As a result, resistance to antibacterials could become an increasingly significant factor in determining old-age mortality in the future.

6. Projecting the Future

6.1 *Introduction*

6.1.1 The first part of Section 6 discusses, in broad terms, how mortality is likely to change in the future. The latter part of the section discusses methods of mortality projection and investigates the impact of using different assumptions for future change.

6.1.2 It is highly probable that in the first few decades of the 21st century, mortality rates for elderly people in the U.K. will improve at a faster rate than ever before. Life expectancy at retirement will surge upwards. The 'cohort effect' has been a wave of rapid improvements, rippling upwards through mortality rates. For the past four decades, people born between 1925 and 1945 have benefited from faster mortality improvements than those born in adjacent generations. Now, people born in this generation have reached, or are close to reaching retirement age. The 'cohort effect' is poised to shift mortality rates for elderly people downwards, drastically altering our expectation of post-retirement lifespan.

6.1.3 More specifically, it is likely that mortality improvements in the first decade of the 21st century will be greatest for people in their 60s and 70s. In the second decade, they will be greatest for people in their 70s and 80s, and so on. As a result, mortality forecasts using projections of improvement rate by year-of-birth (such as the interim basis published by the C.M.I. Bureau in 2002), rather than attained age, will probably be most accurate.

6.2 *Evidence Backing the Prediction of Rapid Mortality Improvement at Older Ages*

6.2.1 Some of the evidence supporting the conclusion that life expectancy at retirement age is likely to improve at a faster rate than ever before is summarised below.

— During the past four decades, mortality in the U.K. has improved most rapidly for people born in the period 1925 to 1945. This 'cohort effect' has been observed in mortality rates for the general population of England and Wales (for both men and women) and in data for male assured lives. The cohort effect has also applied, independently, to mortality rates from all the major health-related causes of death. The implication is that the trend is a robust, deep-rooted effect that is highly likely to be projected into the future.
— Mortality rates from smoking-related causes, such as lung cancer, are higher for elderly men and women today than they were in the 1960s. Past trends in cigarette smoking prevalence mean that deaths from these causes are set to fall rapidly at the highest ages.
— Aside from the particular influence of the cohort effect, there has been a general 'ageing of mortality improvement.' The rate of mortality improvement has been accelerating at older ages. This trend has been observed in the U.K. and in a range of developed countries around the world.
— Rates of mortality from cancer and circulatory disorders are steadily falling. There appears no sign that these trends are slowing. Together these causes of death account for approximately two thirds of all deaths for people in England and Wales aged 70 and above.
— The Government White Paper, published in 1999, *Saving lives: our healthier nation* (Department of Health, 1999) introduced new targets for reduced rates of future mortality. The targets, which previously applied to people aged *under 65*, now also include people aged between 65 and 75. This means that the Government is either becoming increasingly focused on the health of elderly people, or believes that mortality is likely to improve significantly for this age group.
— Life expectancy at age 65 in the U.K. is currently low relative to other developed countries. Despite the recent improvements, mortality from heart disease in the U.K. is amongst the worst in the world and cancer survival rates are poor relative to other countries in Europe. There is, therefore, plenty of potential for improvement.
— Countries with evidence of similar 'cohort effects' to the U.K. have seen rapid improvements being projected well into old-age. For instance, in Japan the fastest rates of improvement for females are presently in the 80 to 89 year age range.
— Medical advances are occurring at an accelerating pace. Scientific developments, such as the decoding of the human genome, have the potential to yield increasingly more significant gains in life expectancy.
— Finally, mortality rates are highest at older ages. Therefore, the potential for improvement is the greatest. It is thought by some (Turner *et al.*, 1998) that medical technology — such as cancer treatment and surgical procedures for heart disease — could be used to a far greater extent to reduce mortality at older ages.

6.3 *The Pace of Improvement for the 1925-45 Cohort*

6.3.1 It is very likely that future rates of mortality improvement in the U.K. will continue to be high, and possibly the highest, for people born in the period 1925 to 1945. It is more difficult to say whether future rates of improvement for people born in this period will be higher or lower than the rates they are currently experiencing.

6.3.2 The *current* rate of improvement for a man born in the early 1930s (at the epicentre of the cohort effect) is between 3.5% p.a. and 4.0% p.a. The long-term average for a man born in the early 1930s, calculated over the period since the 1960s, is between 2.5% p.a. and 3.0% p.a. In favour of increased rates of improvement are:

— observations of an 'ageing' of mortality improvement (Section 2.18); and
— the accelerating pace of scientific and medical advances (Section 4).

In favour of reduced rates of improvement are:

— evidence that part of the cause for the cohort effect has been changes in smoking prevalence, a behaviour change that cannot be repeated in the future (Section 2.20); and
— the fact that causes of death become increasingly correlated with advanced age, making mortality improvements 'harder work' (Section 4).

6.3.3 In the SIAS paper 'Mortality in the Next Millennium' (Willets, 1999) the following statement was made.

> "On balance, it seems more likely that mortality improvements (for specific years of birth) will reduce from their present levels to lower long-term values (say from 3.3% p.a. to 2.5% p.a. ...). However, you could argue that this really is quite a pessimistic view. If you were valuing pension or annuity liabilities then maybe a 'prudent best estimate' would be to assume no reduction from current rates of improvement ...?"

6.3.4 It is notable that, since the publication of this paper, the pace of mortality improvement for the 1925-45 cohort has not reduced. In fact, the reality has been quite the opposite. In Section 2.7 it was noted that improvements for the 'healthy cohort' over the past five years have been significantly higher than those over the previous five.

6.4 *Improvements for Younger Adults*

6.4.1 The general focus of this section is on mortality improvement for older people. However, some brief observations are made on the likely pattern of improvement at younger ages.

6.4.2 Future mortality improvements for younger adults are subject to considerable uncertainty. It is difficult to predict whether rates are more likely to rise or fall. Younger adults — say those in their 20s, 30s and early 40s — have:

— benefited most (in relative terms) from the reductions in infectious disease mortality during the last century and are therefore most vulnerable to re-emergence of disease and antibacterial resistance;
— experienced very limited mortality improvements in the 1980s and 1990s and even seen deteriorations at some ages;
— experienced the greatest number of deaths from the AIDS and vCJD epidemics; and
— experienced substantial increases in mortality during the 1990s from a number of other causes, including accidental deaths, drug and alcohol abuse, liver disease, epilepsy and infectious diseases (other than AIDS).

6.5 *Differentials by Social Class*

6.5.1 Section 2.22 described how social class differentials have widened in recent decades. As a result, the mortality experience of assured lives and annuitants has improved more rapidly than that of the general population.

6.5.2 Present day social and political structures appear unlikely to *drastically* reduce wealth inequalities. Wealth and health are becoming increasingly correlated, with mortality improvements driven by 'lifestyle' and medical technology. This suggests that future mortality improvements for insured lives are likely to continue to outstrip those for the population as a whole. However, this is an area of considerable uncertainty and the arguments for and against greater mortality differentials are highly subjective.

6.5.3 With this uncertainty, it may be more reasonable to use the past as the best guide to the future. For the purpose of projecting future mortality rates for annuitants and pensioners it is also more prudent to assume social class differentials continue to widen.

6.6 *Methods of Mortality Projection*

6.6.1 Many methods have been used for projecting future rates of mortality, and there are a number of useful review papers and books which discuss the various methodologies. Good references for this topic include:
— Mortality on the move (Benjamin & Soliman, 1993);
— Mortality change and forecasting: how much and how little do we know (Tuljapurkar & Boe, 1998);
— Forecasting mortality in developed countries: insights from a statistical, demographic and epidemiological perspective (edited by Tabeau *et al.*, 2001); and
— National population projections: review of methodology for projecting mortality (GAD, 2001).

6.6.2 The GAD review classified potential methods of projecting mortality rates into three essential types:

— process-based methods which concentrate on the factors that determine deaths and attempt to model mortality rates from a bio-medical perspective;
— explanatory-based methods which employ a causal forecasting approach, for instance using variables such as economic or environmental factors; and
— extrapolative methods based on projecting historical trends in mortality into the future.

6.6.3 In practice, extrapolative models tend to be favoured by the vast majority of demographers and actuaries. Extrapolative models can be split into:
— parametric models which involve fitting a parameterised curve (or surface) to data for previous years and then projecting these parameters forward;
— targeting methods which involve interpolating between current mortality rates and a set of target rates, which are assumed to hold at a certain future date; and
— trend methods which involve the projection of historical trends into the future.

All of these methods can either be deterministic or stochastic and can either model aggregate rates of mortality or trends in individual causes of death (which are then combined to produce aggregate projections). All models include a significant element of subjective judgement.

6.7 *The Method Employed by the Government Actuary's Department*

6.7.1 Official population projections for the U.K. and constituent countries are produced by the GAD, usually every two years. The latest projections, the '2002-based projections', were published in December 2003 (GAD, 2003). The current method of projecting mortality involves setting target rates of mortality improvement by age and gender for the 25th year of the projection (for the 2002-based projections, the target rates were 1.0% at all ages for males and females in 2027). Base mortality rates and the annual rates of improvement by age and gender for the first year of the projection are estimated from an analysis of past trends. These improvement rates are assumed to converge (exponentially) to the target rates over the first 25 years of the projection. After the 25th year, the target rates of improvement are assumed to halve every further 25 years.

6.7.2 Future mortality rates are obtained by applying the projected rates of mortality improvement by age and calendar year successively to the assumed base rates of mortality. This is done on a year-of-birth basis for generations born before 1947 (to allow for the 'cohort effect') and on an attained-age basis for those born since 1947.

6.7.3 A number of variant projections are also made using alternative mortality improvement scenarios.

6.8 *Methods Adopted by International Agencies*

6.8.1 There is a wide variety of methodologies for projecting mortality adopted by developed countries and by organisations such as the United Nations. A few examples are given in the following paragraphs.

6.8.2 A recent review by Eurostat (Cruijsen & Ding, 2002) of the latest projections carried out by 29 EEA and EU candidate countries found two broad approaches to forecasting mortality. The first was to extrapolate death rates, life expectancy at birth or other summary indicators. The second was to set a medium or long-term 'target level' for the model parameters used and then to interpolate between the latest observations and the targets to produce annual rates. Most countries used the second approach, sometimes combined with the use of a model life table. Some countries used other forecast techniques such as 'extrapolation of a function' or constant mortality. One country used graphical extrapolation and another used an explanatory model. Many countries reported using expert opinion for making mortality assumptions and many looked at mortality trends in other countries. The inclusion of additional information from health survey reporting or official plans or targets was less common. A few countries considered the impact of factors such as education level and income on future mortality levels.

6.8.3 The U.S. Census Bureau 1999-2100 projections use target model life tables for 2150 constructed using information from a variety of sources, including life-expectancy tables for all males and all females for 2065, produced by Lee-Carter stochastic time-series models. These, together with expert opinion on the relative speed of decline in mortality rates of age groups 0 to 14 and 15 to 64 to that for ages 65 and over for the time periods 1990 to 2020 and 2021 to 2150, were then used to derive age- and sex-specific death rates in 2150. Projected death rates for intervening years are then calculated by interpolation between the 1990 base mortality rates (derived from census data) and the assumed 2150 rates. The death rates thus obtained for years 1999 to 2020 were then replaced by a set of rates calculated by interpolating between death rates derived from deaths and population estimates for 1996 to 1998, and the death rates projected for 2021. Death rates for people aged 85 and over are not obtained from deaths and population estimates, but are derived using a model developed by Coale and Kisker, with parameters changed to give death rates of 100% of remaining lives at age 115 for males and females.

6.8.4 The U.S. Social Security Administration (SSA) also produces population projections. Here the trends in mortality rates are analysed by cause of death and three alternative sets of ultimate annual percentage reductions by age, sex and cause of death were determined for 2026 and

thereafter based on consideration of expected medical and environmental changes. Rates of reduction between those derived for the base year of the projections and for 2026 were obtained by a logarithmic formula.

6.8.5 A similar approach is used in mortality projections for the Canadian Pension Plan, but with the target improvements being run in over a 20-year period. The ultimate rates of reduction assumed are the same as those used for the U.S. SSA projections, adjusted to allow for historical differences in Canadian and U.S. experience.

6.8.6 Mortality rates for the 2001-2050 projections for Japan were obtained using an adaptation of the Lee-Carter model, which decomposes mortality rates into the average shape of the surface over time, the general change over time and the pattern of deviations from the age profile as the general level of mortality changes over time and an error term.

6.9 *C.M.I. Bureau Projections*

6.9.1 In 1999 the C.M.I. Bureau published a mortality projection basis for pensioners and annuitants for use with the '92 series' mortality tables. The projection basis made the following core assumptions:

— There are limiting minimum rates of mortality that vary by age and by sex.
— Mortality rates decrease to their limiting values by exponential decay.
— A given percentage of the total future fall occurs in the first 20 years. The percentage again varies by age.
— The same factors apply for 'amounts' and 'lives' based projections and for men and women.

6.9.2 Tables 6.9.2a and 6.9.2b show the exact assumptions made. The projections assumed that mortality rates for younger pensioners would fall dramatically, whereas the improvements at older ages would be more limited; with no improvements at all for people aged 110 and over.

6.9.3 Table 6.9.2b shows that the reduction to the ultimate rate of

Table 6.9.2a. Limiting rates of mortality in the C.M.I. Bureau projection basis for the '92 series' tables

Age	Limiting value as a % of the 1992 base rate
60	13%
70	30%
80	48%
90	65%
100	83%
110	100%

Source: C.M.I.R. 17 — Linear interpolation applies between the values for age 60 and age 110

Table 6.9.2b. Percentage of the total fall that is projected to occur in the first 20 years in the '92 series' C.M.I. projection basis

Age	% of total fall occurring in the first 20 years
60	55%
70	50%
80	45%
90	39%
100	34%
110	29%

Source: C.M.I.R. 17 — Linear interpolation applies between the values for age 60 and age 110

mortality was expected to occur relatively slowly. The precise formula used to produce 'reduction factors' (for age x, year Y) was as follows:

$$\mathrm{rf}(x, Y) = A + (1 - A) \times (1 - B)^{(Y-1992)/20}$$

where:

A = limiting mortality rate for age x as a % of 1992 rate
B = % of the total reduction in mortality rate for age x occurring in the first 20 years.

The reduction factors are applied to the base table to produce mortality rates for future years as follows:

$$q(x, Y) = \mathrm{rf}(x, Y) \times q(x, 1992)$$

where:

$q(x, Y)$ = mortality rate for age x in year Y.

6.9.4 An interim basis for adjusting the '92 series' projections for cohort effects was published in a C.M.I. Bureau Working Paper in 2002. The Working Paper noted the existence of a cohort trend, centred on a slightly earlier generation than seen in U.K. population experience. Data for male assured lives provided clear evidence of year of birth related effects in mortality improvement, with the cohort centred on births in 1926 the most pronounced.

6.9.5 The Working Paper also described an analysis of data for male life office pensioners, retiring at or after normal retirement age. A similar cohort effect was noted in this data set, with the peak improvements also occurring in the 1926 cohort.

6.9.6 These cohort trends were incorporated into future mortality projections outlined in the paper. The mortality projections assumed that the 'width' of the generation experiencing rapid improvement would reduce

with time. For the period 1992 to 2000 the width was taken to be 33 years (i.e. those born between 1910 and 1942). It was then assumed to reduce linearly to one year (i.e. 1926) by the end of the 'cohort period.' The 'cohort period' was taken as being 10, 20 and 40 years for 'short cohort', 'medium cohort' and 'long cohort' projections respectively.

6.10 *The C.M.I. Bureau Mortality Projections Working Party*

6.10.1 The C.M.I. Bureau mortality sub-committee has formed a Working Party to consider the issue of mortality projections. The members of the Working Party are Angus Macdonald (chairman), Adrian Gallop, Keith Miller, Rajeev Shah, Stephen Richards and Richard Willets (most of whom are also co-authors of this paper). During 2003 this group researched methods of mortality projections and aim to produce a Working Paper on the topic early in 2004. This paper will be available to download from the actuarial profession's website.

6.10.2 The C.M.I. Bureau Working Party's report provides a comprehensive review of the issues surrounding mortality projections. In particular it addresses whether C.M.I. Bureau projections should:

— use aggregate or cause-of-death data;
— use population or C.M.I. data;
— project trends by year of birth (cohort) or age; and
— employ a deterministic or stochastic methodology.

6.10.3 It is not necessary to repeat the content of the paper; however, it is worth noting that the Working Paper places considerable emphasis on the uncertainty involved in making a projection. This uncertainty manifests itself in fitting a statistical model to past data and also in the choice of the most appropriate projection model.

6.10.4 A core theme of this paper is that future projections should be grounded in as good an understanding of the past as possible. An understanding of past trends can also help to assess the reasonableness of projection models. The distribution of scenarios produced by a statistical model may be theoretically correct, but either too wide or too narrow given an understanding of the fundamental nature of what is being modelled and the forces shaping it.

6.10.5 Lack of understanding increases uncertainty. However, it should be stressed that even a complete understanding of the past would not allow the future to be predicted with certainty.

6.11 *Some Sample Annuity Rates*

6.11.1 In order to assess the impact of different future scenarios, some sample level annuity rates have been calculated. Tables 6.11a and 6.11b show how much greater the cost of a single life annuity is using various different mortality bases relative to PA(90) − 2 years. PA(90) − 2 years has been selected

as a base comparison because it is probably the heaviest mortality table for annuitants in use. It is currently the standard basis prescribed under the M.F.R. regulations, used to measure the funding position of U.K. pension schemes, which are soon to be replaced. For reference, annuity rates have also been calculated using age adjustments of −4 and −6 years to the PA(90) tables.

6.11.2 Annuity costs have also been derived using more recently published standard tables. The base table for 1979-1982 experience (PMA/PFA80) is still used by some, so rates have been calculated using this table, projected to the year 2010. Likewise, the '92 series' table is also used in practice, so annuity rates have been derived using this basis, projected to the year 2020 (i.e. PMA/PFA92c2020).

6.11.3 The double-entry version of the '92 series' pensioners tables, with future mortality rates derived using the basis prescribed in C.M.I. Report 17 (1999) has also been used (and is denoted PMA/PFA92). In 2002 the FSA announced that life assurers' illustrations of future pension benefits should include annuity rates derived using this basis. This basis is also used for statutory money purchase illustrations contained in annual pension benefit statements for AVCs and defined contribution scheme benefits issued after April 2003 in accordance with TM1.

6.11.4 The PMA/PFA92 tables have also been used with future rates of mortality taken from the interim C.M.I. Bureau projections published in 2002. The suffixes '*sc*', '*mc*' and '*lc*' refer to the 'short', 'medium' and 'long' cohort projections.

6.11.5 The latest GAD basis — the '2002-based' projection published in December 2003 — is also used. Future rates of improvement in the GAD basis for England and Wales have been applied to the medium cohort C.M.I. projection to 2002 (i.e. PMA/PFA92c2002*mc*). In other words, the C.M.I. '92 series' pensioners table, with the C.M.I. medium cohort adjustment applied, was used to produce a set of mortality rates applicable to 2002. GAD improvement rates were then applied to this set of mortality rates to produce a two-way table and used to calculate the values shown in the line labelled 'GAD 2002.'

6.11.6 The 'Benchmark A' basis takes experience from PMA/PFAc1999*mc* (i.e. the life office pensioners table projected to the year 1999 using the medium cohort C.M.I. projection) and then assumes future improvements are in line with the recent experience for the population of England and Wales, with trends projected by year of birth. In other words, current improvement rates by year of birth are assumed to continue indefinitely.

The 'Benchmark B' basis is the same as 'Benchmark A' except that it assumes that rates of improvement will be 25% higher than recent experience for the population (reflecting the fact that mortality improvements for higher socio-economic classes, assured lives and pensioners have been higher than those for the total population for a sustained period).

Table 6.11a. Cost of level annuities payable monthly in arrears for males aged 60 and 65 using various mortality bases in excess of the cost derived using PA(90)M −2 years

	Commencement in 2004				Commencement in 2019			
	2.5% interest		5.0% interest		2.5% interest		5.0% interest	
	Age 60	Age 65	Age 60	Age 65	Age 60	Age 65	Age 60	Age 65
PA(90) −2 years	0%	0%	0%	0%	0%	0%	0%	0%
PA(90) −4 years	6%	7%	5%	6%	6%	7%	5%	6%
PA(90) −6 years	12%	14%	10%	11%	12%	14%	10%	11%
PMA80c2010	7%	6%	6%	5%	7%	6%	6%	5%
PMA92c2020	21%	21%	17%	18%	21%	21%	17%	18%
PMA92	20%	18%	16%	15%	24%	24%	20%	20%
PMA92*sc*	26%	26%	20%	22%	30%	31%	24%	26%
PMA92*mc*	28%	29%	22%	24%	32%	34%	25%	28%
PMA92*lc*	33%	36%	25%	29%	37%	41%	28%	33%
GAD 2002	30%	32%	23%	26%	34%	37%	26%	30%
Benchmark A	38%	39%	27%	30%	43%	49%	30%	36%
Benchmark B	44%	47%	31%	34%	51%	60%	35%	43%

Table 6.11b. Cost of level annuities payable monthly in arrears for females aged 60 and 65 using various mortality bases in excess of the cost derived using PA(90)F −2 years

	Commencement in 2004				Commencement in 2019			
	2.5% interest		5.0% interest		2.5% interest		5.0% interest	
	Age 60	Age 65	Age 60	Age 65	Age 60	Age 65	Age 60	Age 65
PA(90) −2 years	0%	0%	0%	0%	0%	0%	0%	0%
PA(90) −4 years	5%	6%	4%	5%	5%	6%	4%	5%
PA(90) −6 years	11%	13%	8%	10%	11%	13%	8%	10%
PFA80c2010	3%	3%	3%	3%	3%	3%	3%	3%
PFA92c2020	10%	11%	8%	9%	10%	11%	8%	9%
PFA92	9%	9%	7%	7%	13%	13%	9%	10%
PFA92*sc*	14%	15%	10%	12%	17%	19%	12%	15%
PFA92*mc*	15%	17%	11%	13%	18%	21%	13%	16%
PFA92*lc*	20%	23%	14%	17%	23%	27%	16%	20%
GAD 2002	17%	20%	12%	15%	20%	24%	14%	18%
Benchmark A	20%	22%	13%	15%	24%	28%	16%	20%
Benchmark B	24%	26%	15%	18%	30%	35%	19%	24%

6.11.7 The methodology used to derive the rates of improvement for the Benchmark A and B bases was to use log linear regression to calculate annualised rates of improvement for all individual ages from 20 to 89 using England and Wales population data for the period 1993 to 2001. The central year was therefore 1997 and a year of birth was notionally assigned to each rate of improvement using this central year. So for age 50, for example, the notional year of birth was 1947. The improvement rate for each year of birth

was then further smoothed by taking a 5 year rolling average. So for 1947 the smoothed rate was the average rate from 1945 to 1949. For years of birth before 1910, a simple extrapolation method was used to determine the rates of improvement, with 0.5% p.a. taken as the minimum rate of improvement for the oldest lives.

6.11.8 The method adopted was basic, but this is not important as the reason for the exercise was simply to provide a comparison basis for the other mortality projections. It is not suggested that the most likely future scenario will be one in which the 'current' pace of mortality improvement continues indefinitely. However, it is interesting to use it as a benchmark. Table 6.11c contains the full set of improvement assumptions used.

6.11.9 The results shown in Tables 6.11a and 6.11b are striking. For pension schemes the most relevant figures are for annuities commencing in the future, so the annuities commencing in 15 years time are of most interest. There is a very large difference between annuity rates calculated using different bases. Notably, the increased cost of moving from PA(90)M -2 years to PMA92u2019*mc* using a 2.5% interest rate for annuities for males aged 65 commencing in the year 2019 is 34%. PMA92u2019*mc* is equivalent in annuity cost terms to PA(90)M with a 12-year age deduction. The 'Benchmark B' basis shows an even greater increased cost (i.e. 60% for males aged 65). The equivalent age deduction to PA(90)M required to produce the same cost is 19 years. The same cost can also be derived using PA(90)M -2 years with an interest rate deduction of 195 basis points (applied to both pre- and post-retirement interest rates).

6.11.10 There are also significant differences in annuity values for current commencement using different varieties of the cohort basis. The difference between the long and short cohort bases is over 5% for males and females aged 65 using a 5% interest rate. This is certainly significant given the profit margins generally declared on annuity business. The difference between the short cohort C.M.I. basis and the 'Benchmark B' basis is greatest for males aged 65 (where the difference is over 10%).

6.11.11 It was worth noting that the C.M.I. projections assume the same rates of improvements for males and females, despite the fact that recent improvements have been higher for men than women. As a result the financial impact of using the 'Benchmark A' or 'Benchmark B' bases relative to the C.M.I. projections is lower for females.

6.11.12 It is also notable that applying the improvements underlying the latest GAD 2002-based projection for England and Wales (GAD, 2003c) to base mortality rates at 2002 derived from the C.M.I. tables with the medium cohort projection, produces annuity values which generally fall somewhere between those produced by the medium and long cohort C.M.I. projections.

Table 6.11c. 'Smoothed current' rates of mortality improvement for the England and Wales population by year of birth

Year of birth	Male	Female	Year of birth	Male	Female
1975	0.9%	0.1%	1937	2.8%	2.2%
1974	0.4%	−0.3%	1936	2.9%	2.4%
1973	0.3%	−0.4%	1935	3.3%	2.6%
1972	−0.1%	0.5%	1934	3.5%	2.9%
1971	−0.2%	1.1%	1933	3.7%	3.1%
1970	−0.2%	1.0%	1932	3.9%	3.1%
1969	−0.3%	1.3%	1931	4.0%	3.2%
1968	−0.3%	1.2%	1930	3.9%	3.3%
1967	−0.4%	1.0%	1929	3.8%	3.2%
1966	−0.4%	0.9%	1928	3.7%	3.0%
1965	−0.1%	1.0%	1927	3.5%	2.8%
1964	−0.1%	0.6%	1926	3.3%	2.5%
1963	−0.1%	0.7%	1925	3.1%	2.3%
1962	0.1%	0.5%	1924	3.0%	2.0%
1961	0.3%	0.4%	1923	2.8%	1.8%
1960	0.5%	0.6%	1922	2.6%	1.7%
1959	0.7%	1.1%	1921	2.6%	1.6%
1958	0.8%	1.4%	1920	2.5%	1.6%
1957	1.0%	1.3%	1919	2.6%	1.6%
1956	1.1%	1.3%	1918	2.6%	1.6%
1955	1.2%	1.2%	1917	2.6%	1.7%
1954	1.2%	1.1%	1916	2.5%	1.6%
1953	0.8%	0.9%	1915	2.3%	1.5%
1952	0.6%	0.9%	1914	2.1%	1.3%
1951	0.5%	1.0%	1913	1.9%	1.2%
1950	0.3%	0.9%	1912	1.7%	1.1%
1949	0.4%	0.8%	1911	1.6%	1.1%
1948	0.9%	1.0%	1910	1.5%	1.0%
1947	1.4%	1.1%	1909	1.3%	0.9%
1946	1.9%	1.5%	1908	1.2%	0.9%
1945	2.3%	1.6%	1907	1.0%	0.8%
1944	2.6%	1.7%	1906	0.8%	0.8%
1943	2.6%	1.7%	1905	0.7%	0.7%
1942	2.6%	1.6%	1904	0.5%	0.6%
1941	2.5%	1.5%	1903	0.5%	0.6%
1940	2.5%	1.7%	1902	0.5%	0.5%
1939	2.5%	1.8%	1901	0.5%	0.5%
1938	2.7%	2.0%	Pre-1901	0.5%	0.5%

6.12 *General Conclusions*

6.12.1 So far the paper has provided a detailed analysis of mortality change in the U.K. and of some of the forces shaping current and anticipated future improvements. The likely course of future mortality change has been discussed and the financial impact of different scenarios explored.

6.12.2 At this point, it is worth making some general comments about future trends in longevity. This paper has stopped short of making an

absolute recommendation of a suitable allowance for future mortality improvement. An appropriate assumption will always depend on the precise characteristics of the group whose mortality is being projected. Furthermore, the publication of an updated C.M.I. projection basis, to be used in conjunction with a new set of standard tables based on 1999-2002 experience, is planned for later in 2004.

6.12.3 However, it has been demonstrated that the financial consequences of adopting different assumptions for future improvement can be very significant. It seems probable that, in some cases, the mortality assumptions currently used by actuaries do not make sufficient allowance for likely future improvement. Further, when considering mortality risk, it is likely that many actuaries do not examine the impact of using a reasonable range of such improvements.

6.12.4 In Section 2 we reflected upon the dramatic changes in mortality seen over the course of the 20th century. Table 2.2a showed that mortality rates at some ages are now just 2% of the rates that applied 100 years ago. This magnitude of change can scarcely have been thought possible at the beginning of the last century. Would our actuarial predecessors have imagined such an improvement was possible?

6.12.5 In Section 4 we discussed the accelerating pace of medical advances and noted that some scientists are predicting enormous extensions in our potential lifespan.

6.12.6 In other words, we have proof that very large changes in mortality can occur over relatively short periods of time and we have predictions of very large change in the future. Given that actuarial advice can relate to periods of over 50 years, what should our reaction be to these two facts? At the very least we should always be keen to ensure that users of actuarial advice are aware of the possibility that substantial increases in longevity are possible in the future. Our advice should be framed with this possibility in mind and we should be aware of the financial implications of rapid mortality improvement.

6.12.7 At the same time, we should also be aware that some demographers argue there are biological constraints which limit the extent to which life expectancy can increase. The potential threat from infectious diseases has also been described. Future increases in longevity are highly probable, but not inevitable.

7. Implications

7.1 *Introduction*

In this final section of the paper some of the implications of anticipated increases in longevity will be discussed. In particular the following areas will be addressed:

— the implications for life assurance companies;
— the implications for final salary pension schemes;
— the implications for general insurers;
— wider social and economic implications; and
— the implications for the actuarial profession.

7.2 *The Implications for Life Assurance Companies*

7.2.1 Actual and anticipated increases in longevity have wide-ranging implications for life assurers. As the focus of this paper is life expectancy in retirement, the implications relating to protection products (though interesting and significant) are not discussed. However, to borrow a quotation by Peter Drucker (1999), also used in the SIAS paper presented by Wadsworth, Findlater & Boardman (2001):

> "By providing financial protection against the major 18th- and 19th-century risk of dying too soon, life insurance became the biggest financial industry of that century [...]. Providing financial protection against the new risk of not dying soon enough may well become the next century's major and most profitable financial industry."

7.2.2 The implications of increased longevity in retirement for life assurance companies include:

— losses being declared on existing annuity books;
— increased reserves for guaranteed annuity options (GAOs);
— increased capital requirements;
— capital being more difficult to raise and reinsurance being more difficult to obtain;
— increased asset-liability matching issues;
— an increased focus on mortality issues from equity analysts and rating agencies; and
— development of alternative annuity products and use of additional rating factors.

In many respects these points are being compounded by the impact of proposed legislation and regulatory change. The points listed above are discussed in turn.

7.3 *Losses on Existing Annuity Books*

7.3.1 If mortality experience improves faster, or is expected to improve faster, than allowed for in pricing, then a mortality loss will be made on a tranche of business. How this impacts on overall profitability depends on how the other experience elements fare, notably investment returns. However, it is clear that adjustments in mortality assumptions have had an impact on the profits declared by life assurance companies in recent years.

7.3.2 An equity analyst's report on the U.K. insurance market (JP Morgan, 2003) listed four companies who had announced increases in annuity reserves in Q1 2003 in excess of £50m as a result of reviewed mortality assumptions. In one case the impact of an insurance company moving to the 'short cohort' projection (i.e. the weakest) described in the interim C.M.I. projections (C.M.I., 2002) was a reduction in profits of £140m.

7.3.3 Many life assurance companies' response to this has been to pull back from the sharp end of the annuity market. The JP Morgan report describes how a number of major life assurance companies have taken a deliberate decision to maintain perceived profitability at the expense of volumes of new business.

7.4 *Increased Reserves for Guaranteed Annuity Options*

7.4.1 One of the most immediate problems for U.K. life insurers lies with their guaranteed annuity options (GAOs). The following graph reproduced from Wilkie *et al.* (2003) shows the dramatic effect of increasing longevity of annuitants in the calculation of reserves for GAOs:

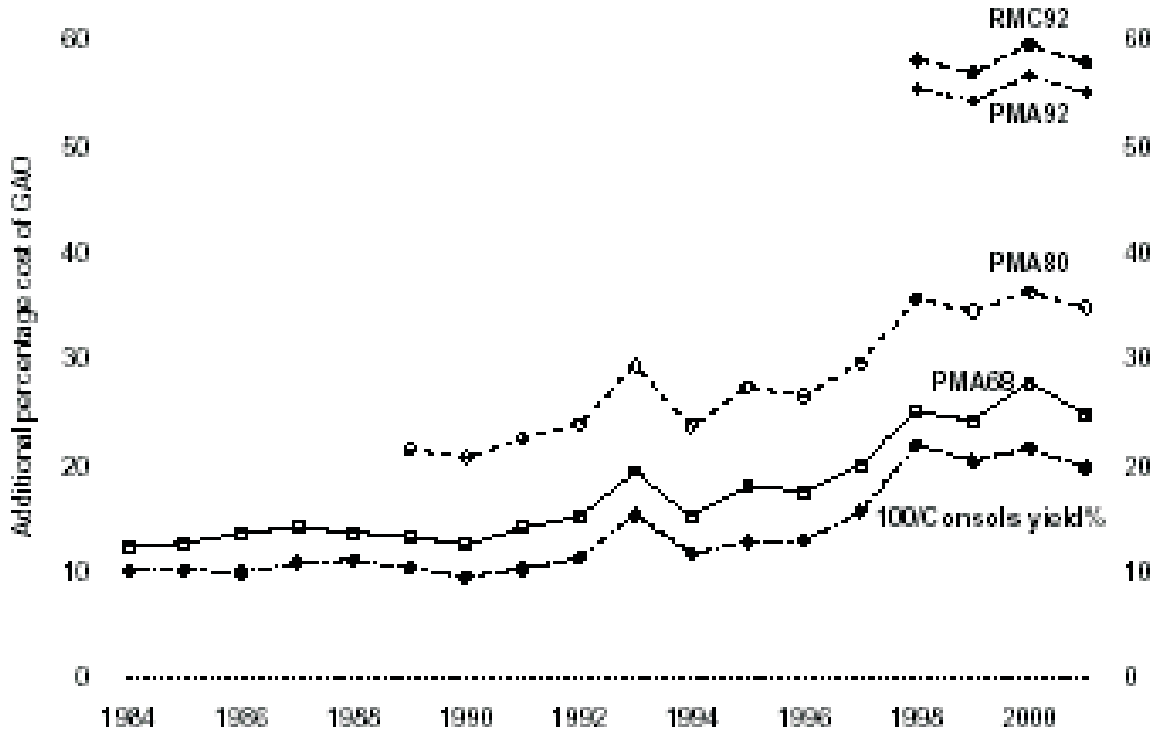


Figure 7.4. 99th quantile of present value of cost of GAO per 100 units of single premium under the 1984 Wilkie investment model; portfolio figures for various life tables from Figure 5.4 in Wilkie *et al.* (2003)

Figure 7.4 shows the percentage additional reserve per unit of single premium. Increasing longevity, as reflected in the successive generations of life tables shown, has dramatically increased the size of the GAO liability reserve. The figures shown are the 99th quantile reserves from 10,000 simulations. For further details see Wilkie *et al*. (2003).

7.4.2 It is notable that Figure 7.4 shows the impact of moving to the '92 series' tables. The effect of adopting the interim C.M.I. projections outlined in the Working Paper published in 2002 would be even more dramatic.

7.5 *Increased Capital Requirements*

7.5.1 The fact that mortality has improved more rapidly in recent years than was generally expected, has highlighted the fact that future mortality projections are subject to considerable variation and uncertainty. This has caused some actuaries (e.g. Willets, 1999) to question the degree of prudence in annuity reserves. There is a case for greater margins for prudence and allowance for potential variation in experience from that expected. Of course, the imminent implementation of the *Integrated Prudential Sourcebook* and the regime of Individual Capital Assessment for insurers will serve to intensify the focus of actuaries on adverse scenarios and adverse deviations in future assumptions.

7.5.2 A recent ABI report entitled *The future of the pension annuity market* (Wadsworth, 2003) suggested that capital requirements for annuity business might increase as a result of the new regulatory framework. Specifically, the report suggested that:

> "... as capital management operated by life companies and sought by the regulator becomes more sophisticated, it seems at least possible that already fairly substantial capital requirements to back annuities will increase, although precise requirements will develop with practice."

7.6 *Difficulties in Raising Capital and Obtaining Reinsurance*

7.6.1 As discussed in Sections 7.3 and 7.5, insurers require more capital, and may have greater capital requirements in the future, in relation to annuity business. At the same time, the likely volumes of future annuity new business, resulting largely from historic patterns of pension sales and changing demographics, may also add to the level of capital required (Wadsworth, 2003).

7.6.2 However, the U.K. life industry is already under capital pressure and may have used up the market's appetite for exposure to life insurers in the form of rights issues and subordinated debt. In any event, the market's appetite for life company debt may be felt to move more in line with equity levels and it is not a reliable, regular source of capital for writing annuity business.

7.6.3 The greater emphasis being put on risk-based approaches by actuaries, management, investors and regulators means that mortality risk will be increasingly evaluated for its impact on the overall capital requirements of a company.

7.6.4 The scale of the issue has increased as a function of the increase in the proportion of life office liabilities that are annuities. For many companies longevity risk is now the second largest risk that the company faces (and in some cases the largest). It is a systematic risk (in that it is likely that if one group of lives survives longer than expected, most of the other groups will as well) and as such it does not easily sit in one financial institution. Systematic risks should either be diversified or lead to substantial capital requirements. It is important to note that while life office shareholders can diversify risks of this nature, policyholders cannot.

7.6.5 The desirability of diversification is beginning to be recognised in the market place by the increasing interest in finding new places for mortality and longevity risk. Examples include the recent Barclays securitisation and the Swiss Re excess mortality bond issued in November 2003 (Swiss Re issued $250m of 'principal-at-risk variable-rate mortality catastrophe-indexed note' with note holders at risk for any deterioration of a defined mortality index, based on population mortality in the U.S.A., France, the U.K., Switzerland and Italy, exceeding 130%). Securitisations linked to unexpectedly high rates of mortality improvement have proved more difficult to place.

7.6.6 Alternative approaches to raising capital for longevity risk, such as securitisation, may have proved difficult since market perception is that insurers are much better placed than market participants in assessing such risks.

7.6.7 Reinsurers have also been reluctant to take on large blocks of longevity risk, although there is a market for limited tranches of annuity business. This may have been the case because there has been a gap in perception between reinsurance actuaries and their life office counterparts as to the correct price to charge for longevity risk. Reinsurers have seen substantial and enduring mortality improvements in relation to protection business and potential medical advances impacting critical illness products. There are generally close relationships between reinsurance actuaries, medical underwriters and doctors and this background naturally helps reinsurers to appreciate the potential impact of future medical advances.

7.6.8 The reinsurance market for longevity risk may grow in future years as the gap in perception reduces through a more widespread appreciation of the potential for future improvements in mortality. However, the pricing of these contracts, and that of the various non-life catastrophe bonds, suggests that the reinsurance cost of mitigating the peak mortality costs is much greater than the marginal benefit of writing more business for many companies. It can therefore be reasonably expected that the costs of buying an annuity will rise faster than the

improvement in mortality and that there will be a concentrated attempt to find new homes for longevity risk.

7.6.9 The ABI paper discussed earlier highlights the potential gulf growing between the demand for annuity products and the supply of capital to write this business.

7.7 *Increased Asset-Liability Matching Issues*

Under-estimating mortality experience has effects beyond the immediate mortality loss. For example, if an insurer had a portfolio of backing assets well matched to liabilities, lighter mortality rates than expected lead to a mismatched position. The assets would then be too short and the insurer is not just suffering from the longevity risk but is also exposed to unexpected additional reinvestment risk. The new framework for capital assessment is likely to lead to more focus on the cash flow matching of annuity portfolios.

7.8 *Increased Focus on Mortality Issues*

7.8.1 Longevity risk is now firmly on the list of questions which ratings agencies and equity analysts routinely ask life insurers. This has been highlighted in reports such as the JP Morgan publication referred to earlier.

7.8.2 As well as focusing on amounts of annuity liabilities and volumes of new business written by insurers, equity analysts and rating agencies are likely to place much more emphasis on how insurers are managing longevity risk. Some insurers and reinsurers have established dedicated teams specialising solely in researching mortality in retirement.

7.9 *Development of Alternative Products in a Changing Retirement Market*

7.9.1 The U.K. retirement income market has seen some important changes in the past decade. Whilst growth in annuity purchases has continued, the market has become far more diverse than the standard non-profit annuity differentiated by age and gender. With-profits and unit-linked annuities have given people a greater degree of investment choice and the advent of the impaired life and enhanced annuity has given people with poorer life expectancy a correspondingly higher annuity rate (around one-tenth of the annuity market is now of the enhanced or impaired type). Furthermore, since the legislation introducing income drawdown in 1996, many people have been able to delay having to purchase any kind of annuity (to age 75) by leaving their funds invested, but drawing a non-annuitised income.

7.9.2 The SIAS paper 'Reinventing Annuities' (Wadsworth, Findlater & Boardman, 2001) proposed a new unit-linked annuity structure with 'survival bonuses' added to fund values each year. The product sought to combine the flexibility and investment advantages of drawdown with the protection

against 'mortality drag' afforded by a traditionally annuity. One major life office has developed and launched such a product.

7.9.3 Many of the product developments have been caused, at least partially, by trends in increasing life expectancy. Life assurance companies have sought to minimise their exposure to longevity risk through with-profits and alternative product designs. Customers have responded well to product designs that provided them with enhanced income levels to compensate for falling annuity rates. Rates have fallen largely because interest rates have fallen, but also because of increasing life expectancy in retirement.

7.9.4 The remainder of this section discusses some additional features and issues faced by annuity providers in more detail.

7.9.5 Logically, the existence of impaired life and enhanced rate annuities must be causing anti-selection effects for writers of standard annuities. This subject was explored in depth by Ainslie (2000), who suggested that significant anti-selection effects in the standard annuity experience would appear once the impaired/enhanced annuity market reached its current size. It may be too early to tell if this is the case from C.M.I. data. In any case there are socio-economic differentials to complicate the matter: the larger the fund, the more likely the annuitant is to seek the specialist retirement income advice which may result in an impaired/enhanced annuity. Any anti-selection phenomenon is likely to be confined to insurers' open-market option business. Coincidentally, it is also the holders of larger funds who are more likely to either buy a with-profits annuity, or else go into income drawdown instead of buying a non-profit annuity of any kind. These socio-economic associations may well have had the effect of offsetting part of any anti-selection since, as discussed in Section 2, it is the wealthiest annuitants who live longest.

7.9.6 The distinction generally made to differentiate 'impaired' and 'enhanced' annuities is that impaired annuities are aimed at people with serious medical conditions and are rated according to individual assessments by medical underwriters or doctors. On the other hand, enhanced annuities are products which offer different annuity rates based on various objective rating factors which are linked to life expectancy, such as smoking status, socio-economic class, geographical location and other indicators of health. The potential 'impaired' market is perhaps 10% of the total, whereas the potential enhanced market is maybe 90%. This statement is made on the basis that life expectancy in retirement will be greatest for wealthy, professional, non-smokers who are in perfect health, drink alcohol in moderation, are neither under- nor over-weight and live in the South of England. The vast majority of annuity purchasers do not have all of these characteristics. A significant challenge for annuity providers in the near future will be how they adapt to the growth of the enhanced sector.

7.9.7 While most annuities are currently written to the under-65s, change can be expected in the age profile of annuity buyers over the next few years. At some point the growth in income drawdown as an alternative to annuity purchase will slow down, or even reverse. Recent stock market falls will make some drawdown customers seek the security of an annuity instead of taking further investment risks. Even those who do not want to annuitise their funds will have to do so by age 75, when annuitisation is compulsory. Drawdown first became an available option in 1996, so any 65-year-olds who went into drawdown then are now only two or three years away from compulsory annuitisation. Compulsory annuitisation looks set to stay, following the Government's killing of a Private Member's Bill in Summer 2003 which would have removed the limit at age 75.

7.9.8 From April 2005, a long-overdue simplification of the tax regime surrounding pensions is likely to come into force (HM Treasury/Inland Revenue, 2003). Some of these changes, whilst introduced in the name of simplification, will nevertheless change the annuity market and therefore the nature of the longevity risk in that market. The simplest change — that of increasing the commutation limit to £14,000 (in 2005) — could possibly remove a large proportion of the very small funds from the annuity market. Many people will be tempted to take the (taxed) fund instead of using it to buy a very small annuity. This would change the longevity risk as it is primarily the less-well-off who have small funds and would be tempted to take the cash if their marginal tax rate is low. Longevity in the U.K. is starkly differentiated by socio-economic group, and any sharp reduction in shorter-lived annuitants would increase life offices' longevity risk, for both internal-vesting and open-market annuities.

7.9.9 At a time when insurers face considerable challenges in controlling their longevity risk, possible regulatory changes simultaneously threaten to take away some of their existing flexibility. An E.U. proposal to forbid the use of gender as a rating factor in insurance products would have important consequences for the annuity market. We noted in Section 2.2 that life expectancy at age 65 in the population of England and Wales is currently 16.0 years for men and 19.1 years for women. Unisex annuity rates are already a reality for protected rights pension funds, but they would be a new and challenging development for annuities purchased from other pension funds.

7.9.10 From the perspective of potential customers of post-retirement products, some of these developments are not good news. Taken together, future increases in life expectancy, increasing awareness of the risk of providing longevity insurance in a low inflationary environment, changes in legislation and shortages in market capacity and capital, may well lead to worsening annuity rates. Products which seek to minimise the longevity risk for insurance companies, such as with-profit annuities, could become a better value option for customers.

7.10 *The Implications for Final Salary Pension Schemes*

7.10.1 The longevity liabilities on the balance sheets of U.K. insurers amount to several tens of billions of pounds, with much of this held in with-profits funds. Yet these liabilities are dwarfed by the equivalent total for U.K. private sector defined benefit schemes. Indeed, the estimated total *deficits* of defined benefit schemes have amounted to many billions of pounds in recent times. In January 2004 Hewitt, Bacon & Woodrow (2004) estimated that the total deficit in FTSE100 companies' balance sheets was around £50 billion (substantially less than some figures quoted by commentators early in 2003).

7.10.2 The implementation of FRS 17, an accounting standard which discloses such pension deficits on corporate balance sheets, has been delayed but not abandoned. Ratings agencies claim always to have considered pension scheme risks and deficits when assessing the credit-worthiness of companies. Nevertheless, the advent of deficit disclosure under FRS 17 does seem to have led company executives and ratings agencies to show much greater interest in the risks associated with defined-benefit pension schemes, particularly where the pension scheme is large relative to the sponsoring employer's own balance sheet. Initially, this heightened interest was focussed on the deficits arising from recent equity volatility. However, interest has begun to shift towards the longevity risk being borne by final salary schemes.

7.10.3 In order to assess the impact of likely increases in life expectancy on the finances of pension schemes, it is necessary to know what mortality bases are currently being used in practice. It is unfortunate that FRS 17 has no requirement to disclose the mortality assumptions used. In fact it is difficult to find published information describing the mortality bases used by pension schemes for accounting or funding purposes. The lack of publicly available information was discussed by O'Brien (2003) who suggested disclosure of a form of 'pension cost index' constructed to allow comparison of mortality assumptions between schemes. A simpler alternative would be a requirement to disclose sample life expectancy values implied by the choice of mortality basis (for instance, life expectancies for males and females attaining age 65 now and in 15 and 30 years' time).

7.10.4 The figures given in Tables 6.11a and 6.11b show that the cost of providing an annuity, and hence a pension at retirement age, varies dramatically according to the mortality basis assumed. The likely mortality of pension scheme members will obviously vary according to industry and location. For instance, pensioners from a financial services company in Bournemouth would have a very different life expectancy in retirement to pensioners from a heavy engineering firm based in Glasgow. However, the continuing reference to PA(90) -2 years in the (soon-to-be-replaced) MFR basis is hard to understand given the extent of mortality improvements since the construction of this table (derived from mortality experience from the period 1967-1970) and anticipated future improvements.

7.10.5 A review of the MFR basis published in 2000 (Faculty of Actuaries and Institute of Actuaries Pensions Board, 2000) indicated that, in order to maintain the strength of the basis when introduced, a move to using PA(90) −4 years was required. In 2001 the Pensions Board recommended that the dividend yield assumption be further adjusted, partly to reflect additional improvements in mortality (Faculty of Actuaries and Institute of Actuaries Pensions Board, 2001). The changes to the MFR basis introduced in Spring 2002 retained the minus two-year rating but modified the change to the dividend yield introduced at the same time to make allowance for improving mortality. It could then be argued that the current MFR basis makes implicit allowance for a minus four-year age rating.

7.10.6 Given the figures shown in Tables 6.11a and 6.11b it is certainly not clear that PA(90) with a four-year rating would now be an appropriate basis (even for a 'heavy mortality' scheme such as one for manual workers with relatively small pensions).

7.10.7 The Pensions Board has started to sponsor the collection and analysis of mortality experience of self-administered pension schemes by the C.M.I. Bureau. This development is very welcome as actuaries should also be clear in demonstrating that they have allowed for likely increases in life expectancy.

7.10.8 In a lecture to the Actuarial Profession on 2 September 2003 Adair Turner, chairman of the Pensions Commission, made the following comment:

> "It does seem that the actuarial profession was slow to respond to the accumulating evidence of the 1980s and 1990s, which have seen rapid reductions in mortality rates in older age groups, and for far too long has allowed its forecasts for the future to be influenced by the much slower mortality progress of the 1960s and 1970s, and has therefore worked on a limit-to-life and rectangularisation hypothesis. Since 1980 life expectancy forecasts have consistently undershot emerging reality, and even today I suspect that the latest published forecast from GAD and C.M.I. understate the best estimates of future life expectancy, and will be revised up further in the next public set of figures."

7.10.9 Employers sponsoring final salary schemes are making promises to their employees that extend up to 70 or 80 years into the future. This paper has sought to show that substantial increases in life expectancy are likely in the future, and some scientists are claiming that we will be seeing the fruits of anti-ageing research within decades. Actuaries should be clear in spelling out to employers and trustees the nature of the risks behind the promises they are making.

7.10.10 It is interesting to note that in two recent lectures to the profession the need for more flexible definitions of 'retirement age' was highlighted. Adair Turner discussed the possibility of not fixing retirement age in advance, but instead using a formula which (for example) said "the retirement age in the scheme is currently X, but this will increase in each five

years by two thirds of any increase in forecast life expectancy at age 65 calculated by the GAD.''

7.10.11 In a lecture to the Faculty of Actuaries on 6 October 2003 Tom Kirkwood, Professor of Gerontology at Newcastle University, outlined a very similar idea. He suggested that increases in life expectancy in retirement should be split into two components: an increase in retirement age and an increase in post-retirement life expectancy (a 'life rise').

7.10.12 If final salary schemes are to continue to play a significant role in pension provision in the 21st century, more flexible definitions of retirement age may become an essential component of scheme design.

7.10.13 The remainder of Section 7.10 discusses some additional issues that actuaries advising pension schemes are facing and how these issues interact with the mortality trends we have discussed.

7.10.14 Changes are proposed to the Limited Price Indexation (LPI) basis after April 2005. The LPI basis currently increases a pension in line with the Retail Price Index (RPI) with a cap of 5% per annum. The proposal would reduce this cap from 5% to 2.5%, which would lower the cost of providing LPI benefits and also the costs of buying out those benefits with an insurer.

7.10.15 In the context of mortality improvements and longevity, escalating annuities such as LPI benefits contain significantly more longevity risk than non-escalating benefits. This is due to the escalation rate partially offsetting the effect of the interest rate used in discounting future payments. Reductions to the escalation rate, such as the LPI-related measure described above, would not only decrease the cost of the benefit, but would also reduce the longevity risk exposure (to say nothing of the reduction in reinvestment risk) in defined-benefit pension schemes.

7.10.16 Another change applies to solvent employers choosing to wind up a defined-benefit pension scheme on or after 11 June 2003. In such circumstances, the cost of buying full benefits for all members has to be met. 'Buying full benefits' refers here to a so-called 'bulk buy-out' or 'bulk Section 32 transfer' to an insurance company. On the face of it, this might increase the demand for bulk buy-outs.

7.10.17 However, the market for bulk buy-outs in the U.K. has a number of unique characteristics which may affect employers. The bulk buy-out market in the U.K. is currently only served by a small number of insurance companies with specialist expertise. Bulk annuities are also capital intensive contracts, yet U.K. life insurers do not have as much capital as they had three years ago. Insurers are also particularly wary of the extra longevity risk in the deferred annuities which commonly sit in defined-benefit schemes. This tends to result either in conservative pricing for the deferred annuities in a wind-up, or else a refusal to take schemes where the deferred pensioners exceed a prescribed proportion of liabilities. A result of this is that some schemes may not be able to buy out at any bearable price. If the

demand for bulk buy-outs is to expand, these features of the bulk market point more towards an increase in buy-out costs than to an expansion of supply. Employers wanting to rid themselves of the longevity liabilities in their pension scheme may even find no takers at any price.

7.10.18 In past years, future longevity was not considered to be key risk factor for pension schemes. The existence of a high inflation, high investment return environment served to dampen down the impact of future increases in life expectancy. At the beginning of the 21st century it is clear that longevity is now a major issue.

7.11 *The Implications for General Insurers*

7.11.1 Increasing longevity has only recently had a direct impact on general insurers, who are now bearing the cost of accident victims living for long periods of time and receiving expensive benefits (notably medical costs). In the past such benefits were valued somewhat arbitrarily in the U.K. and the courts did not take mortality rates into account explicitly. Consequently, longevity trends were not a major issue.

7.11.2 The introduction of the Ogden Tables to determine actual awards changed that. The Actuarial Tables with explanatory notes for use in Personal Injury and Fatal Accident cases (more commonly referred to as the Ogden Tables) provide an aid for those assessing the lump sum appropriate as compensation for a continuing future pecuniary loss or consequential expense, or cost of care in court cases involving personal injury or fatal accident. The tables set out various multipliers, which are applied to an assessment of future annual loss or expense to obtain a present capital value.

7.11.3 Although these tables were first produced in 1984, it was not until the passing of the Civil Evidence Act (1995) that the Ogden Tables were taken to be admissible in evidence for the purpose of assessing, in an action for personal injury, the sum to be awarded as general damages for future pecuniary loss.

7.11.4 The multipliers allow for mortality and take into account the possibilities that the claimants will live for different periods. The mortality assumptions used relate to the general population for England and Wales. The first edition used the mortality rates from the English Life Tables No 13. The tables were updated following the issue of English Life Tables No 14 and No 15. The third edition also introduced a second set of multipliers which were based on the projected mortality rates underlying the 1996-based national population projections for England and Wales. The current, fourth, edition provides a set of multipliers calculated on English Life Tables No 15 mortality rates and a set based on the projected mortality rates underlying the 1998-based population projections for England and Wales. A fifth edition is planned which will use the mortality rates from the 2002-based population projections for England and Wales. In all cases, adjustments to the

multipliers may be made in cases involving impaired lives (for example by adjusting the assumed age of the claimant). It is believed that all judges use the tables based on projected mortality rates in court cases involving the use of the Ogden Tables.

7.11.5 In general, most awards for damages in these cases will ultimately be paid by insurance companies. The accepted use of the Ogden Tables in court cases involving personal injury and fatal accidents, and the continued updating of the Tables to incorporate projected improvements in mortality, suggests that damage awards are likely to increase in the future through assumed increasing longevity. It is important that insurers allow adequately for likely improvements in mortality when setting premiums for business which can give rise to such claims (such as employer's liability and motor). For large claims it can take several years before the case reaches court and the Ogden Tables may have undergone several revisions from those which were current at the date the premiums were set.

7.11.6 In reality improving mortality and medical knowledge has a three way impact. Firstly, more people survive the immediate accident and so become a more expensive claim. However, this is relatively straightforward to monitor and incorporate into premium rates that can be revised annually. Secondly, improving medical knowledge has so far been translated into more expensive treatment. It can be many years before a claim is finally taken to court and improving (but more costly) medical treatments may well have a material impact on the claim. Thirdly, longer life means that the claim will last for very much longer if not settled for cash. This is the area where there is the most impact. It is geared in that most claims are linked to at least the rate of inflation, and often a higher rate, so the result is very sensitive to mortality assumptions.

7.11.7 The impact of increasing longevity is mitigated by the fact that personal injury claims tend to number only a small proportion of total claims. As a result there is a diversification element in the risks faced by general insurers. Mortality is likely to be of more concern to general insurance actuaries in the future than it has been in the past.

7.11.8 There are two areas where there is likely to be a greater impact. The first is where reinsurers are providing excess of loss cover on an account where there are many material personal injury claims. Here the profitability of the account will be very sensitive to mortality experience. The second is the trend to consider the use of structured annuities to be provided by a specialist carrier. The mortality issue and the corresponding concentration of risk may well discourage this trend, even though it may be desirable for many other reasons.

7.12 *Wider Social and Economic Implications*

7.12.1 The implications of increasing longevity for society is a vast topic

and this paper does not seek to cover this area in any detail. It is a subject that the Social Policy Board, and in particular the Ageing Population Group, has been looking at in some depth. In 2002 the Ageing Population Group organised a two-day conference to discuss issues surrounding the ageing population and a further event is planned for 2005. The collaboration between the Actuarial Profession and the International Longevity Centre is also proving to be a very positive development.

7.12.2 On a global level, the demographic shift towards a more elderly population is being seen as one of the major challenges facing the world in the 21st century. A report published by the Center for Strategic and International Studies (C.S.I.S.) in the U.S. (2003) summarised the situation as follows:

> "Today, there are 30 pension-eligible elders in the developed world for every 100 working-age adults. By the year 2040, there will be 70. In Italy, Japan and Spain, the fastest ageing countries there will be 100 ...Ten years ago, global ageing barely registered as a policy issue. Today, with the retirement of the large postwar baby-boom generations looming just over the horizon, it is the focus of growing concern among political and policy leaders worldwide."

7.12.3 In the countries most vulnerable to rising old-age dependency costs (France, Italy and Spain according to the C.S.I.S. report) changing demographics are already having a significant social and political impact. Notably in Italy, organisers claimed that 1.5 million people attended a rally in Rome in December 2003 to protest against pension reform. The Italian government wants to increase the pension contribution period from 35 to 40 years, to encourage Italians to work to 60 or 65. Many currently retire on full benefits at 57 (BBC News Online, 6 December 2003).

7.12.4 The severity of the issue in the U.K. is not thought to be as great. However, this is partially due to the relatively low level of the U.K. Basic State Pension and its continued link to the Retail Price Index. From a political perspective, it is debatable whether state pension benefits as a percentage of average earnings can continue to reduce in future years, especially given the increasing age of the electorate.

7.12.5 It is not difficult to find examples of how the ageing population and increased forecasts of post-retirement longevity affect public finances. Section 7.10 referred to an estimated deficit of £50 billion in the balance sheets of FTSE100 companies in respect of defined benefit pension schemes. By definition, this is an unfunded pension promise (or, more accurately, a yet-to-be-funded pension promise). However, even this is overshadowed by the £350 billion figure revealed to Parliament in April 2003 by the financial secretary, Ruth Kelly. This figure is in respect of unfunded public-sector defined-benefit pensions, calculated according to the FRS 17 standard to be applied to private companies. These liabilities have been estimated by the GAD as follows:

Table 7.12a. Unfunded public-sector scheme liabilities (source: GAD) and all tax receipts excluding National Insurance contributions (source: Inland Revenue)

Year	Liability in £bn	Tax receipts in £bn	Tax receipts As percentage of liabilities
1987	124	112	90
1988	139	121	87
1989	154	132	86
1990	169	141	83
1991	184	144	78
1992	201	143	71
1993	228	148	63
1994	235	164	70
1995	265	178	67
1996	284	190	67
1997	309	212	69
1998	327	227	69
1999	345	242	70
2000	367	256	70

Despite several years of overall tax rises, the unfunded public-sector pension liabilities have grown faster than tax receipts.

7.12.6 Bigger still are the implicit liabilities in the unfunded state pension benefits for old age: the basic state pension, the minimum income guarantee, SERPS and the state second pension (S2P). To put these figures in perspective, the U.K. gross domestic product at market prices in 2002 was £1,044 billion (source: O.N.S., http://www.ons.gov.uk).

7.12.7 As mentioned earlier, official population projections for the U.K. and constituent countries are produced by the GAD, usually every two years. These projections in turn form the basis for a range of others carried out by government, including projections of marital status, household projections, labour force projections, subnational projections and numbers in education; in all of which it is assumed that the numbers of people will aggregate to the totals assumed in the national projections. These various projections are widely used for planning and policy decisions. Although projected mortality is only one component of population projections (along with fertility and migration), it is important that the projections are seen to use credible assumptions concerning future mortality rates, especially in the area of National Insurance.

7.12.8 The latest Quinquennial Review of the National Insurance Fund (GAD, 2003b) projects future income and expenditure up to the year 2060/61. Over this period, the projected number of pensioners is largely determined by the assumptions about future mortality and the current age structure of the population. Mortality assumptions have relatively little impact on future numbers of working age.

Table 7.12b. Effect of variant mortality projections on projected joint Class 1 pay-as-you-go contribution rate required to balance income and expenditure in the year, excluding the contributions allocated to the NHS, based on real earnings growth of 2% p.a. with price uprating

	2001-02	2010-11	2020-21	2030-31	2040-41	2050-51	2060-61
Principal projection	19.1%	18.4%	16.4%	16.4%	15.4%	14.9%	14.9%
				Mortality variants			
'Low' improvement	0.0%	−0.1%	−0.3%	−0.5%	−0.8%	−0.9%	−0.9%
'High' improvement	0.0%	+0.1%	+0.3%	+0.5%	+0.8%	+0.9%	+1.0%
Constant improvement	0.0%	+0.1%	+0.3%	+0.6%	+1.0%	+1.5%	+2.0%

Source: G.A.D. (2003b)

7.12.9 Although the main results of the review are produced using the principal population projection, the review also provides a sensitivity analysis showing the results of varying the underlying assumptions. In the case of mortality, support ratios and changes in the contribution rates required are shown using the variant mortality projections which are published together with the principal population projections.

7.12.10 A potential driving force in the area of public policy is public awareness or otherwise as to how long they might actually live, especially after retirement and what the cost of providing retirement incomes, medical and other long term care and so on is likely to be. It is likely that most people will underestimate life expectancy at age 65, especially given the widespread misapprehension that life expectancy at a given age is life expectancy at birth less that age. The 'Benchmark A' projection basis, described in Section 6.11.6., was used to derive the probability of survival to age 90 for a male and a female aged 65 in 2004. The survival probabilities were 47% and 55% respectively.

7.12.11 Increasing public awareness of the implications of longevity may well lead to changes in working patterns, increasing retirement ages and phased-in retirement and pressure for the provision of some form of protection against outliving assets and the risk of poverty in extreme old age. Indeed the old age pension scheme as originally constituted was more concerned with insurance against longevity than providing retirement income.

7.12.12 Another factor which will have an impact on public policy is the extent to which increases in longevity are increases in healthy life expectancy. If the increases are accompanied by increases in ill-health, then there is likely to be increased demand on the health service and the providers of care.

7.12.13 However, the over-riding implication of the anticipated increases in life expectancy is that people will remain in work for longer in the future. The age at which people retire will inevitably have to increase and this trend

will necessarily drive changes in all aspects of our society. Our attitudes towards work, education, health, our families and saving for the future will all change as we live longer. As actuaries we have a vital role in helping to inform the wider debate.

7.12.14 If we are to maintain the confidence of professionals in other disciplines, it is imperative that actuaries seek to participate at the cutting edge of research into longevity trends. We should be striving to offer fresh insights and developing new theories of mortality change.

7.13 *The Implications for the Actuarial Profession*

7.13.1 Mortality is an important subject for the actuarial profession. It has played a central role in actually making us a profession, rather than a collection of individuals who work in life assurance and pensions. More research into this area is clearly needed. Increases in post-retirement lifespan could have a dramatic impact in so many respects.

7.13.2 As actuaries we are ideally placed to do this research and in the last few years there has been a resurgence of interest in mortality matters in the profession. The number of conference presentations on mortality improvement or annuity pricing has increased sharply. The C.M.I. Bureau are breaking new ground with their research into cohort trends and it is also notable that a joint C.M.I and GAD seminar on mortality projection (October 2003) sought the input of practitioners in demography, gerontology and genetics. The input of professionals in other fields has also been sought in the work of the Actuaries Panel on Medical Advances, established by the Social Policy Board to research the potential impact of trends in medicine. As mentioned earlier, the Pensions Board has begun to sponsor the analysis of combined self-administered scheme mortality experience.

7.13.3 At the same time, it is notable that the study of mortality matters appears to play a lesser role in the actuarial exams. The removal of very U.K specific topics such as the C.M.I. Bureau Reports and English Life Tables makes sense given the move to a syllabus which focuses more on general concepts than facts, especially in the earlier stages of the exams. However, the importance of the subject to the profession, and to the wider community, suggests that a greater emphasis should be placed on understanding mortality trends. This could be achieved by focusing more on the drivers of mortality change, mortality variation and the management of mortality risk in the specialist Life Assurance and Pensions exams. A further step would be the introduction of an exam in the early stages of the syllabus requiring trainee actuaries to study some basic ideas in demography, health, medicine, epidemiology and gerontology. In other words our student actuaries should be armed with a better understanding of how and why people become ill and how and why they age and die.

7.13.4 Actuaries 'make financial sense of the future.' In doing this we need to have an understanding of finance and investment combined with

knowledge of demographic assumptions. We have an almost unique combination of interests. We are 'financial demographers' and as such we should endeavour to understand the forces driving trends in mortality and longevity in the 21st century.

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APPENDIX 1

RECENT TRENDS BY CAUSE OF DEATH

A1.1 *Introduction*

A1.1.1 The figures in this section of the paper show how mortality from individual causes of death has varied during the 1990s and early years of the new century. In all cases the values plotted are the ratios of observed central mortality rates for the population of England and Wales, for the relevant year, age group and cause of death, relative to the comparative 1989 rates of mortality.

A1.1.2 Sections A1.2, A1.3 and A1.4 cover three of the major causes of death — heart attack, stroke and cancer respectively.

A1.1.3 Section A1.5 looks at the recent trends for violent and accidental deaths, significant contributors to mortality rates at younger ages.

A1.1.4 Section A1.6 sets out the recent trend in respect of AIDS-related deaths.

A1.1.5 Section A1.7 covers pneumonia, a significant component of mortality for the elderly and also illustrates some of the potential problems and distortions inherent even in national data for deaths by cause.

A1.1.6 Finally, Section A1.8 considers two causes of death which have shown markedly adverse trends over the 1990s and which have therefore dampened down mortality improvements, particularly for ages 20 to 49.

A1.2 *Recent Trends in Heart Disease Mortality*

A1.2.1 Table A1.2 shows the relative importance of heart disease as a cause of death in 2001 for the population of England and Wales.

Table A1.2. Deaths from heart disease, cause-specific mortality rate per 100,000 lives and deaths as a percentage of all deaths, England and Wales population, 2001

	Age group						
	20-29	30-39	40-49	50-59	60-69	70-79	80+
Males							
Deaths per 100,000	1	8	47	151	452	1,218	2,843
As % of all causes	1%	6%	19%	25%	27%	26%	22%
Females							
Deaths per 100,000	0	2	9	35	159	596	1,959
As % of all causes	0%	3%	6%	9%	16%	20%	18%

Own figures — data source: O.N.S. (2003)

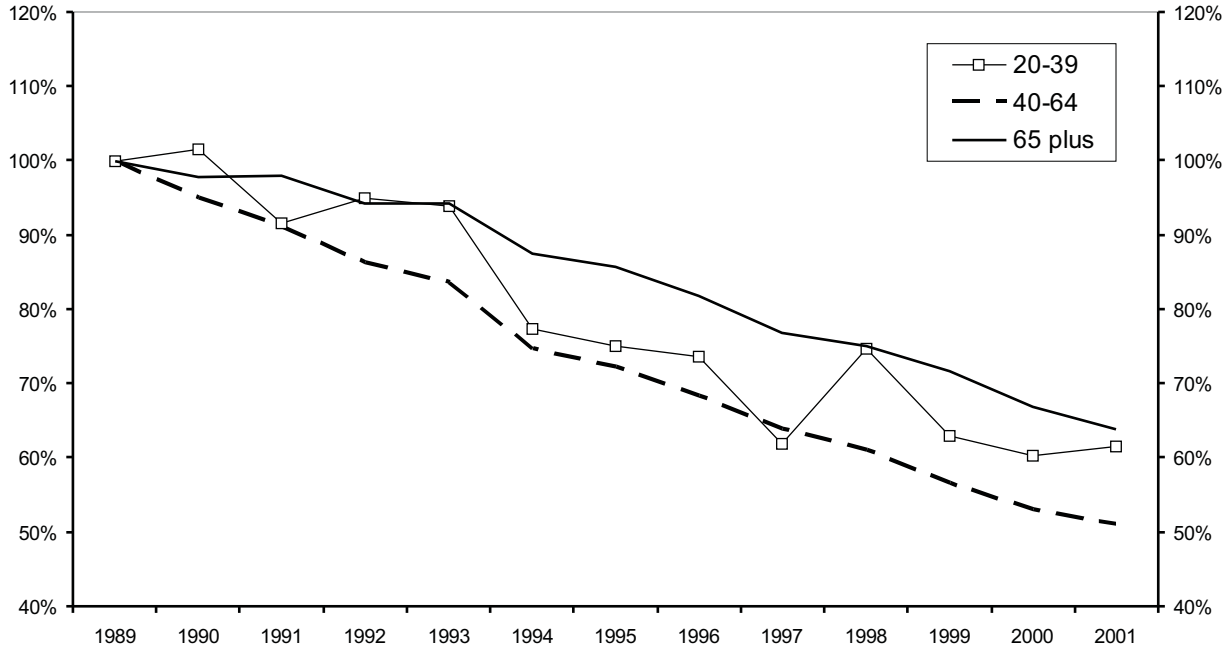
A1.2.2 Figures A1.2(M) and A1.2(F) show that mortality from heart disease has fallen very rapidly and very steadily for men and women of all ages. The smallest fall is for young women, where the small number of deaths has led to significant year-on-year fluctuations in experience.

A1.2.3 The 40-64 age group has seen particularly large improvements. The 2001 rate of heart disease mortality is close to half of the 1989 rate for both men and women — a staggering 50% fall over 12 years! Furthermore, there seems to be little sign of the trend slowing down.

A1.2.4 Some of the factors that help to explain the dramatic reductions in heart disease mortality include:

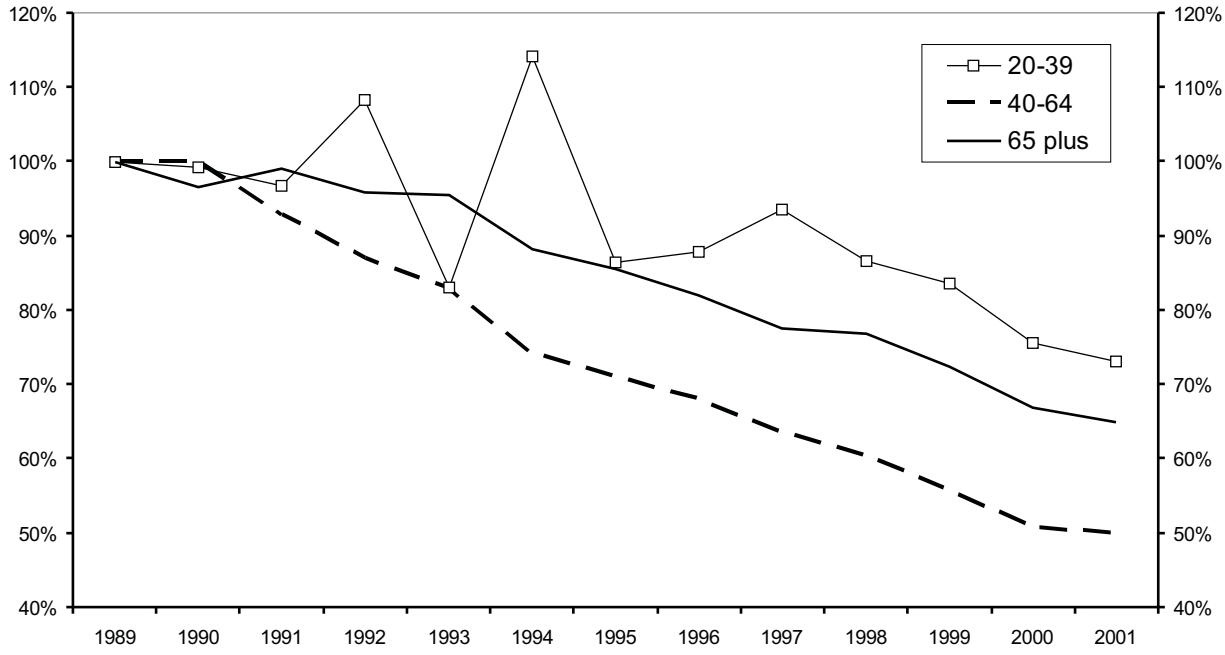
— The reduction in the prevalence of cigarette smoking, which is explored in depth in Sections 2.19 and 2.20. Between 1988 and 2001 the percentage of male adults in Great Britain smoking cigarettes fell from 33% to 28%. The percentage of female smokers fell from 30% to 25% over the same period (O.N.S., 2002).
— Improvements in diet, such as reductions in the consumption of saturated fats. The proportion of food energy taken as saturated fat for adults in Great Britain fell from 17% to 15% between 1988 and 2000. Likewise, the percentage of food energy taken as fats of all kinds fell from 42% to 38% (British Heart Foundation, 2003).
— Reduction in average blood pressure. The mean systolic blood pressure among male adults in England fell from 139 to 136mmHg between 1993 and 2001. The mean for females fell from 136 to 132mmHg (British Heart Foundation, 2003).
— Improved surgical treatments for those suffering from heart disease, both in terms of effectiveness and greater availability. The number of bypass surgery operations in the U.K. doubled between 1989 and 2001 (British Heart Foundation, 2003) and between 1991 and 2001 the number of angioplasties increased almost four-fold.
— The increasingly widespread use of prescription medicines for treating heart disease. Between 1989 and 2000 the number of prescriptions for diseases of the circulatory system in England increased by 238%. Notably over this period the prescription rates for lipid lowering drugs increased 34 times, the prescription rate for anti-platelet drugs increased 10 times, the rate for anti-hypertensive therapy increased 6-fold and prescriptions of nitrates and calcium blockers more than doubled (British Heart Foundation, 2003).

A1.2.5 Deaths have reduced because of both reduced *incidence* of heart disease and improvements in treatment. The relative importance of these two factors is difficult to quantify. However, it is generally believed that changes in incidence have had a more significant impact. Data on incidence is not as robust as that on mortality, but an analysis of the trend in incidence of myocardial infarctions (heart attacks) shows falls over the 1990s of around



Source: own figures based on O.N.S. data (2003)

Figure A1.2(M). Mortality rates from heart disease 1989 to 2001 as a proportion of the rate in 1989, males in England and Wales by age group



Source: own figures based on O.N.S. data (2003)

Figure A1.2(F). Mortality rates from heart disease 1989 to 2001 as a proportion of the rate in 1989, females in England and Wales by age group

35% for both men and women in the 35 to 64 age group (Critical Illness Trends Research Group, 2003).

A1.2.6 It is worth noting that:

— Improvements have not been quite as high for people aged over 65.
— Improvements have been very significant for younger men, but this has had little impact on *aggregate* mortality rates because heart disease only accounts for small proportion of deaths at these ages.

A1.2.7 It is also notable that heart disease mortality has reduced so significantly over a period in which obesity has become far more prevalent. Prevalence of obesity amongst males aged 16-64 in England increased from 7% to 21% between 1986/7 and 2001. The equivalent increase for females was from 12% to 23% (British Heart Foundation, 2003). It is evident that the adverse impact of this trend has been heavily outweighed by the positive factors influencing heart disease mortality. In the future, as this 'epidemic' matures, it may begin to have a more substantial impact, especially on later born generations who will have lived with obesity for longer periods of time.

A1.2.8 In Section 4 a potential development in the treatment of heart disease is discussed, namely the development of a 'polypill' which could, it has been suggested, cut cardiovascular disease by 80%. Given the pace of improvement seen over the last decade, reductions of this magnitude cannot be discounted as unrealistically optimistic.

A1.3 *Recent Trends in Stroke Mortality*

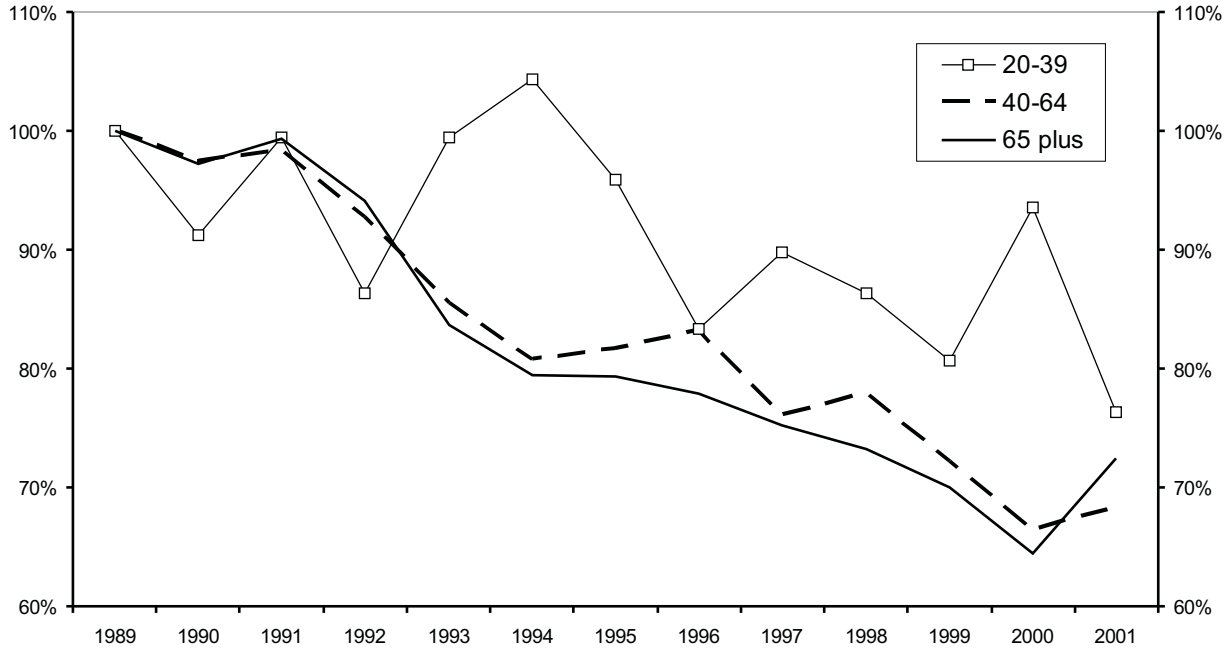
A1.3.1 Table A1.3 shows the relative importance of stroke as a cause of death in 2001 for the population of England and Wales.

A1.3.2 Similar patterns can be seen for stroke mortality as for heart disease mortality — see Figures A1.3(M) and A1.3(F). The experience of

Table A1.3. Deaths from stroke, cause-specific mortality rate per 100,000 lives and deaths as a percentage of all deaths, England and Wales population, 2001

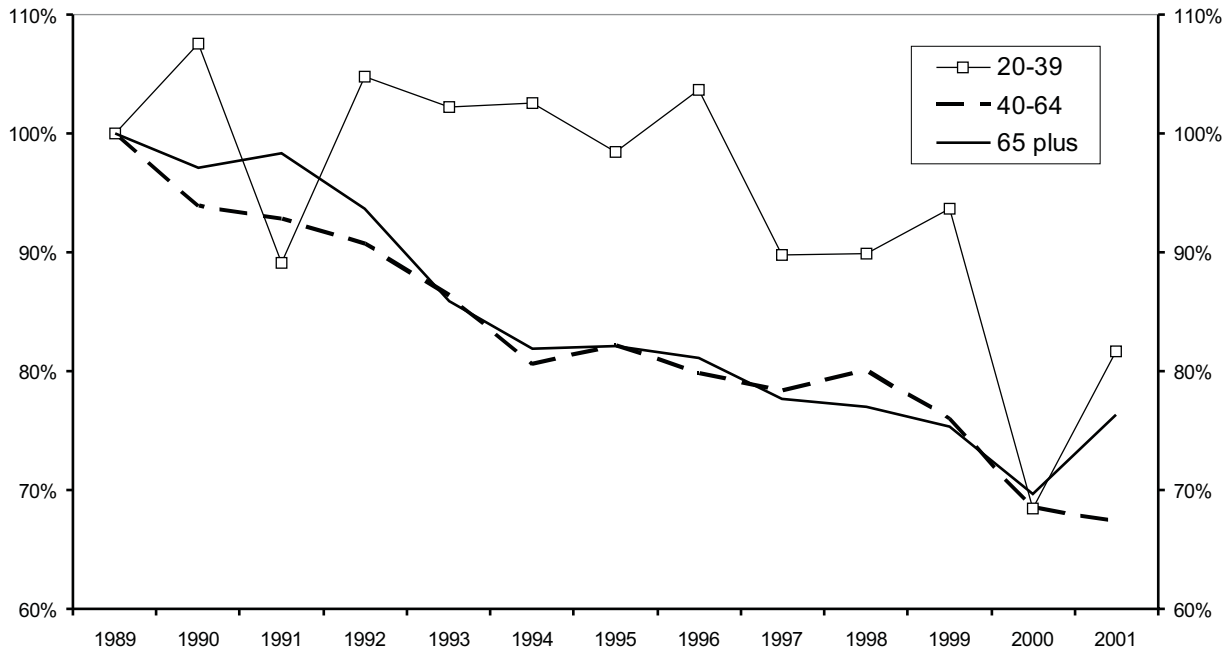
	Age group						
	20-29	30-39	40-49	50-59	60-69	70-79	80+
Males							
Deaths per 100,000	1	3	10	29	97	414	1,590
As % of all causes	2%	2%	4%	5%	6%	9%	12%
Females							
Deaths per 100,000	1	3	9	21	70	338	1,762
As % of all causes	3%	5%	6%	5%	7%	11%	16%

Own figures — data source: O.N.S. (2003)



Source: own figures based on O.N.S. data (2003)

Figure A1.3(M). Mortality rates from strokes 1989 to 2001 as a proportion of the rate in 1989, males in England and Wales by age group



Source: own figures based on O.N.S. data (2003)

Figure A1.3(F). Mortality rates from strokes 1989 to 2001 as a proportion of the rate in 1989, females in England and Wales by age group

younger adults is volatile because of the smaller number of deaths, but for middle-aged and elderly men and women stroke mortality rates have been falling steadily.

A1.3.3 Although still very significant, the rate of fall in stroke mortality rates has been somewhat slower than for deaths from heart disease mortality — around a 30% reduction over 12 years rather than 50%.

A1.3.4 The risk factors contributing to these improvements are similar to those applying to heart disease.

A1.3.5 Again, it is difficult to quantify the relative importance of reduced incidence of cerebrovascular disease and improvements in treatment post-stroke. For strokes, as for heart attacks, data on incidence is not as robust as that on mortality. Analysis of Hospital Episode Statistics for England over the 1990s (source: http://www.doh.gov.uk/hes/) appears to indicate an increase in the recorded incidence rates for strokes for both sexes. However, these figures and trends are likely to have been distorted by changes in diagnostic techniques and criteria. The 'real' incidence of the underlying disease has almost certainly been reducing, although it is clear there have also been significant improvements in stroke mortality due to improved treatment and survival rates.

A1.4 *Recent Trends in Cancer Mortality*

A1.4.1 Table A1.4 shows the relative importance of cancer as a cause of death in 2001 for the population of England and Wales. The table also shows corresponding figures for some specific cancer sites.

A1.4.2 Cancer mortality has not improved at quite the same pace as mortality from circulatory disorders, such as heart disease and stroke — see Figures A1.4a(M) and A1.4a(F). However, improvements have been significant and steady. They have been of a similar magnitude for men and women below the age of 65, but greater for elderly men than women of the same age. This difference stems largely from trends in lung cancer rates.

A1.4.3 Figures A1.4b(M) and A1.4b(F) illustrate the changes in mortality due to lung cancer. For men of all ages, and women in the age groups up to 64, lung cancer mortality rates have been falling steadily. The opposite trend can be seen for women in the 65 and over age group. Since lung cancer is so highly correlated to smoking behaviour this tells us a lot about the impact of cigarette consumption on current rates of mortality improvement. Cigarette smoking is discussed in some depth in Sections 2.19 and 2.20.

A1.4.4 Unlike heart disease, there is no evidence that falling *incidence* rates for cancer have contributed greatly towards reduced rates of mortality. The latest figures for cancer incidence actually show a modest increasing trend (even after systematic data errors are eliminated and temporary increases in incidence due to the introduction of screening programs are allowed for). Also of note is that overall cancer mortality rates have improved more over the 1990s than over the 1970s and 1980s.

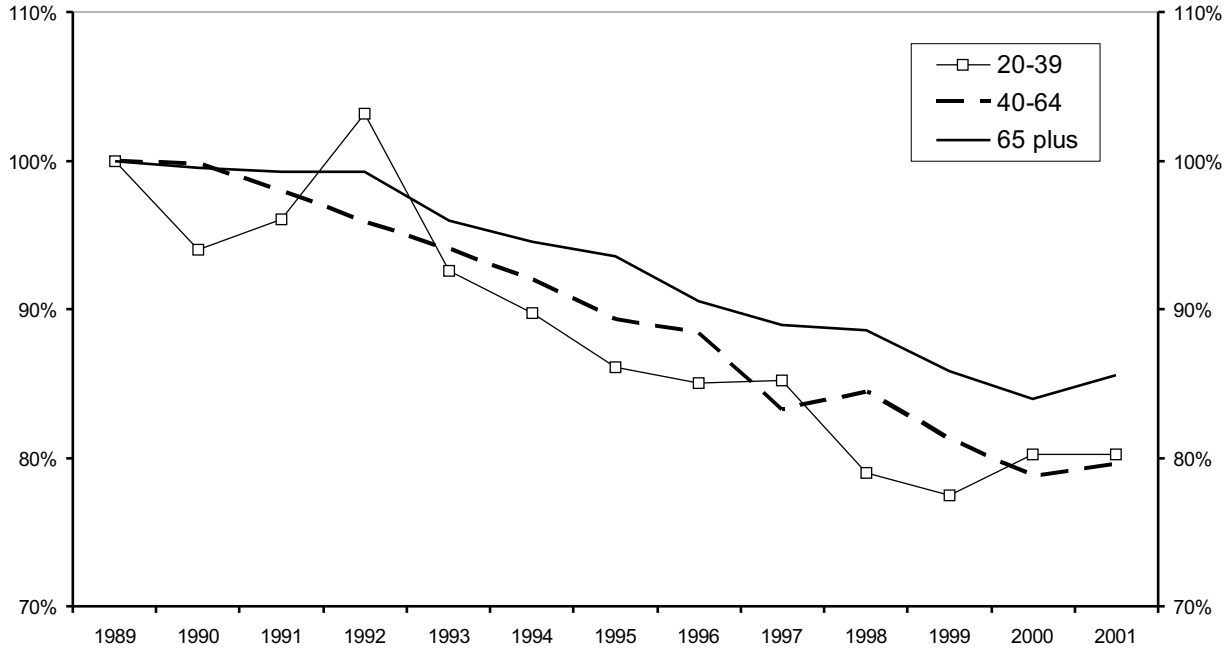
Table A1.4. Deaths from cancer, cause-specific mortality rate per 100,000 lives and deaths as a percentage of all deaths, England and Wales population, 2001

	Age group						
	20-29	30-39	40-49	50-59	60-69	70-79	80+
Males							
Deaths per 100,000							
Prostate cancer	0	0	1	7	49	193	592
Lung cancer	0	1	10	56	183	417	558
Other cancers	7	16	48	164	423	922	1,668
All cancers	7	17	59	227	655	1,531	2,817
As % of all causes							
Prostate cancer	0%	0%	0%	1%	3%	4%	5%
Lung cancer	0%	1%	4%	9%	11%	9%	4%
Other cancers	8%	13%	20%	27%	25%	20%	13%
All cancers	8%	14%	24%	37%	39%	32%	22%
Females							
Deaths per 100,000							
Breast cancer	0	8	25	57	83	127	243
Cervical cancer	1	3	4	5	7	11	14
Lung cancer	0	1	8	36	93	205	196
Other cancers	5	11	37	116	289	621	1,134
All cancers	7	23	74	214	472	965	1,587
As % of all causes							
Breast cancer	2%	14%	16%	15%	8%	4%	2%
Cervical cancer	2%	4%	3%	1%	1%	0%	0%
Lung cancer	0%	1%	5%	9%	9%	7%	2%
Other cancers	17%	18%	23%	30%	28%	21%	10%
All cancers	20%	38%	47%	54%	46%	32%	14%

Own figures — data source: O.N.S. (2003)

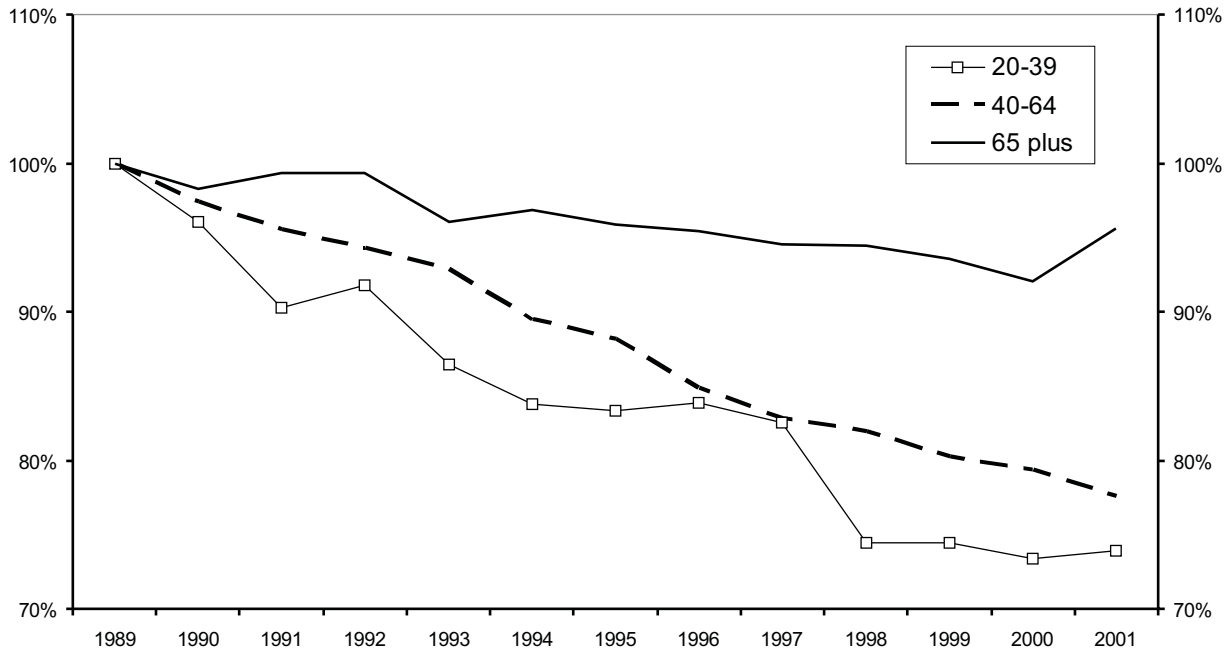
A1.4.5 Mortality from breast cancer has fallen rapidly during the 1990s — see Figure A1.4c(F) — having been broadly level, or slightly increasing, during the 1970s and 1980s. The NHS Screening Programme for breast cancer was initiated in 1988. This Programme has successfully advanced cancer detection amongst the target age group, 50 to 65, and so may well have contributed to falling mortality rates. However, breast cancer mortality rates have fallen by similar degrees for all age groups during the 1990s whilst cancer registrations have risen, suggesting that a major contributor to breast cancer mortality improvements has been advances in treatments for cancer patients.

A1.4.6 Mortality from cervical cancer has also fallen rapidly during the 1990s — see Figure A1.4d(F). However, in contrast to breast cancer,



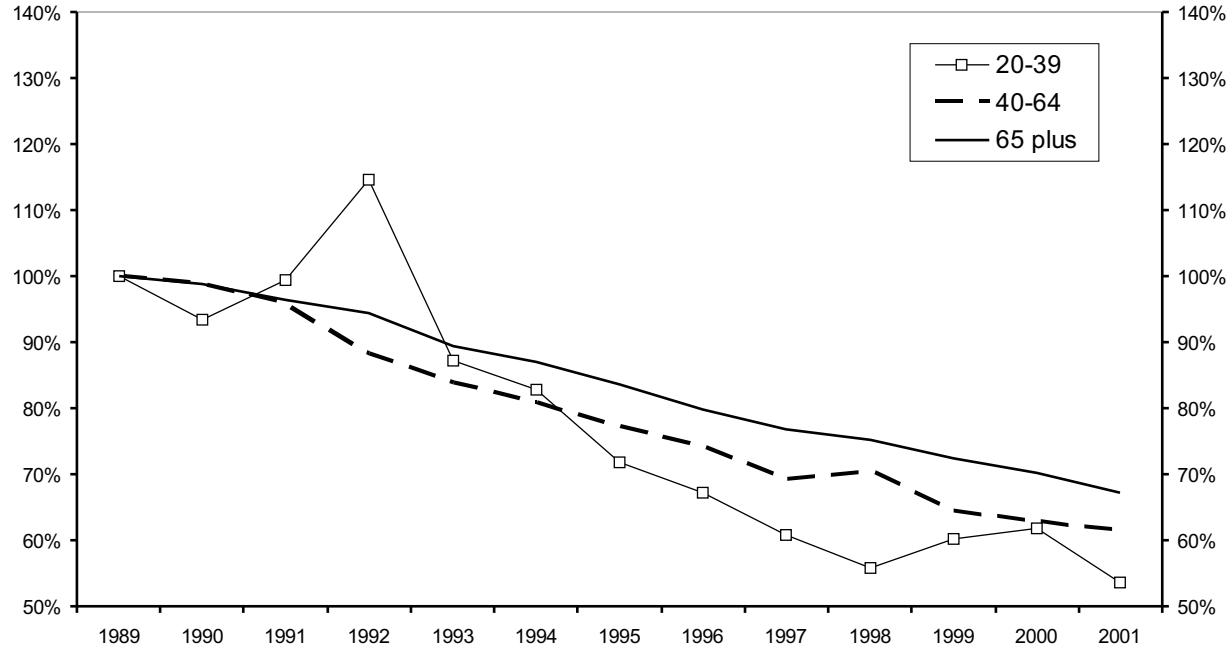
Source: own figures based on O.N.S. data (2003)

Figure A1.4a(M). Mortality rates from cancer 1989 to 2001 as a proportion of the rate in 1989, males in England and Wales by age group



Source: own figures based on O.N.S. data (2003)

Figure A1.4a(F). Mortality rates from cancer 1989 to 2001 as a proportion of the rate in 1989, females in England and Wales by age group

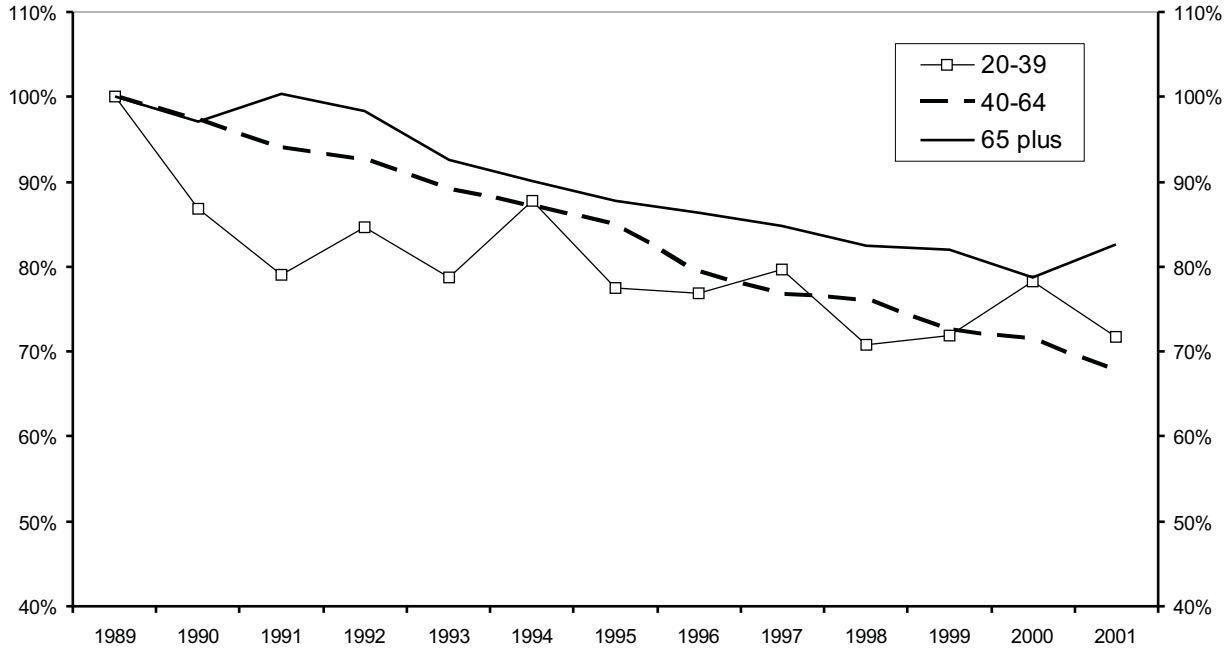


Source: own figures based on O.N.S. data (2003)

Figure A1.4b(M). Mortality rates from lung cancer 1989 to 2001 as a proportion of the rate in 1989, males in England and Wales by age group

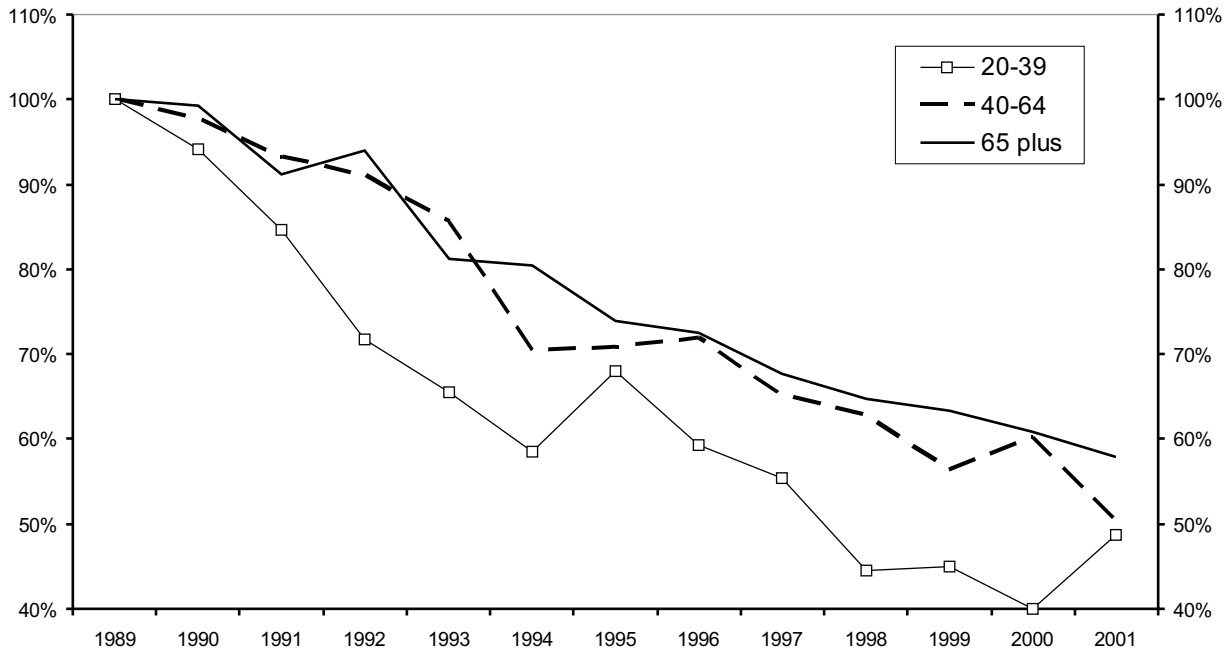
Source: own figures based on O.N.S. data (2003)

Figure A1.4b(F). Mortality rates from lung cancer 1989 to 2001 as a proportion of the rate in 1989, females in England and Wales by age group



Source: own figures based on O.N.S. data (2003)

Figure A1.4c(F). Mortality rates from breast cancer 1989 to 2001 as a proportion of the rate in 1989, females in England and Wales by age group



Source: own figures based on O.N.S. data (2003)

Figure A1.4d(F). Mortality rates from cervical cancer 1989 to 2001 as a proportion of the rate in 1989, females in England and Wales by age group

mortality and incidence rates for cervical cancer have experienced similar changes suggesting that these falls are mainly driven by behavioural changes.

A1.5 *Recent Trends in Violent and Accidental Mortality*

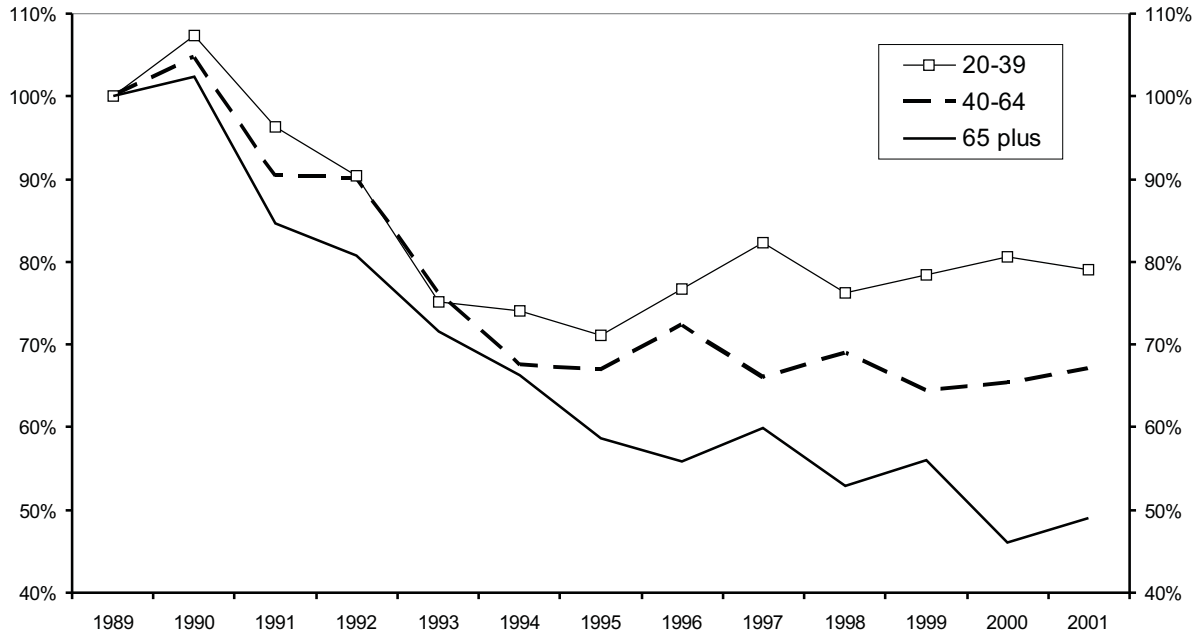
A1.5.1 Table A1.5 shows the relative importance of violence and accidents as a cause of death in 2001 for the population of England and Wales. The table also shows a breakdown of the figures for more detailed categories of cause.

A1.5.2 During the early 1990s mortality rates from motor vehicle accidents fell rapidly, especially for men — see Figures A1.5a(M) and A1.5a(F).

Table A1.5. Deaths from violence and accidents, cause-specific mortality rate per 100,000 lives and deaths as a percentage of all deaths, England and Wales population, 2001

	Age group						
	20-29	30-39	40-49	50-59	60-69	70-79	80+
Males							
Deaths per 100,000							
Motor vehicle accidents	15	11	7	6	6	10	17
Other accidents	11	12	12	12	14	38	170
Suicide	12	16	15	12	9	11	15
Violence	10	10	8	6	5	5	6
All violence and accidents	48	48	42	37	34	63	207
As % of all causes							
Motor vehicle accidents	18%	9%	3%	1%	0%	0%	0%
Other accidents	13%	10%	5%	2%	1%	1%	1%
Suicide	15%	13%	6%	2%	1%	0%	0%
Violence	12%	8%	3%	1%	0%	0%	0%
All violence and accidents	58%	41%	17%	6%	2%	1%	2%
Females							
Deaths per 100,000							
Motor vehicle accidents	3	2	2	2	2	4	8
Other accidents	2	3	5	6	9	30	179
Suicide	2	3	4	4	3	3	5
Violence	3	4	3	3	2	3	5
All violence and accidents	10	11	14	15	16	41	197
As % of all causes							
Motor vehicle accidents	8%	3%	1%	0%	0%	0%	0%
Other accidents	7%	4%	3%	1%	1%	1%	2%
Suicide	8%	5%	2%	1%	0%	0%	0%
Violence	9%	6%	2%	1%	0%	0%	0%
All violence and accidents	32%	18%	9%	4%	2%	1%	2%

Own figures — data source: O.N.S. (2003)



Source: own figures based on O.N.S. data (2003)

Figure A1.5a(M). Mortality rates from motor vehicle accidents 1989 to 2001 as a proportion of the rate in 1989, males in England and Wales by age group

Source: own figures based on O.N.S. data (2003)

Figure A1.5a(F). Mortality rates from motor vehicle accidents 1989 to 2001 as a proportion of the rate in 1989, females in England and Wales by age group

For men in their 20s and 30s this is still one of the greatest single causes of death. So naturally these improvements made significant contributions towards reductions in aggregate mortality at these ages. A number of factors are likely to be behind the trend, including improved car safety features and improved driving behaviour (for example, reduced drink-driving). Disappointingly, in the late 1990s the trend appears to level off for men under age 65.

A1.5.3 Rates of 'other' accidental death mortality (that is, accidents other than motor vehicle accidents) are also most important — in a relative sense — at younger ages. Again, the trend has not been favourable — see Figures A1.5b(M) and A1.5b(F). For men and women aged 20 to 39 the mortality rate for other accidental deaths was higher in 2001 than 1989.

A1.5.4 Violent deaths also form a significant proportion of deaths among younger adults, especially men in their 20s and 30s. During the 1990s the rate of mortality from violence for men aged under age 40 has increased noticeably — see Figure A1.5c(M).

A1.5.5 Suicide mortality — see Figures A1.5d(M) and A1.5d(F) — has fallen during the 1990s for elderly people, but remained broadly level for adults under the age of 40 (where it is a more significant cause of death, in relative terms). The trends are actually more positive than patterns seen during the 1980s, when there were huge increases in the number of suicides among young men.

A1.6 *Recent Trends in AIDS Mortality*

A1.6.1 Table A1.6 shows the relative importance of AIDS as a cause of death in 2001 for the population of England and Wales.

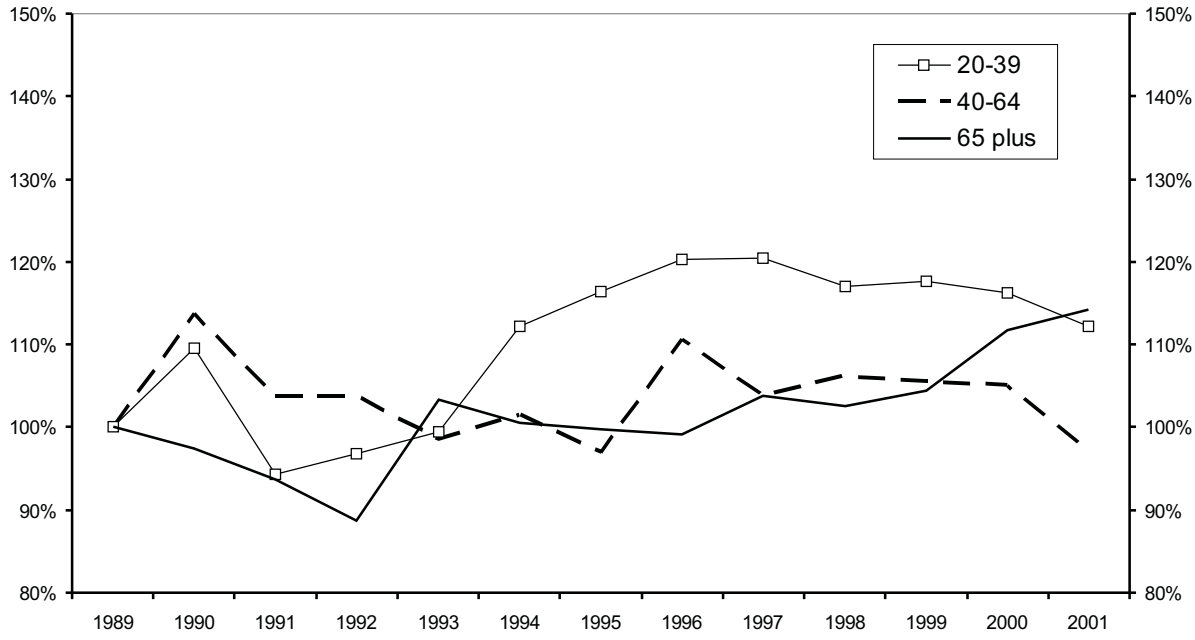
A1.6.2 AIDS currently accounts for a tiny proportion of deaths, even at younger ages. As shown in Figures A1.6(M) and A1.6(F), mortality from AIDS rose steadily during the early 1990s, before falling dramatically over 1995 to 1998, and then levelling off for males but rising again for females. This trend is discussed in detail in A2.2.1.

A1.7 *Recent Trends in Pneumonia Mortality*

A1.7.1 Table A1.7 shows the relative importance of pneumonia as a cause of death in 2001 for the population of England and Wales.

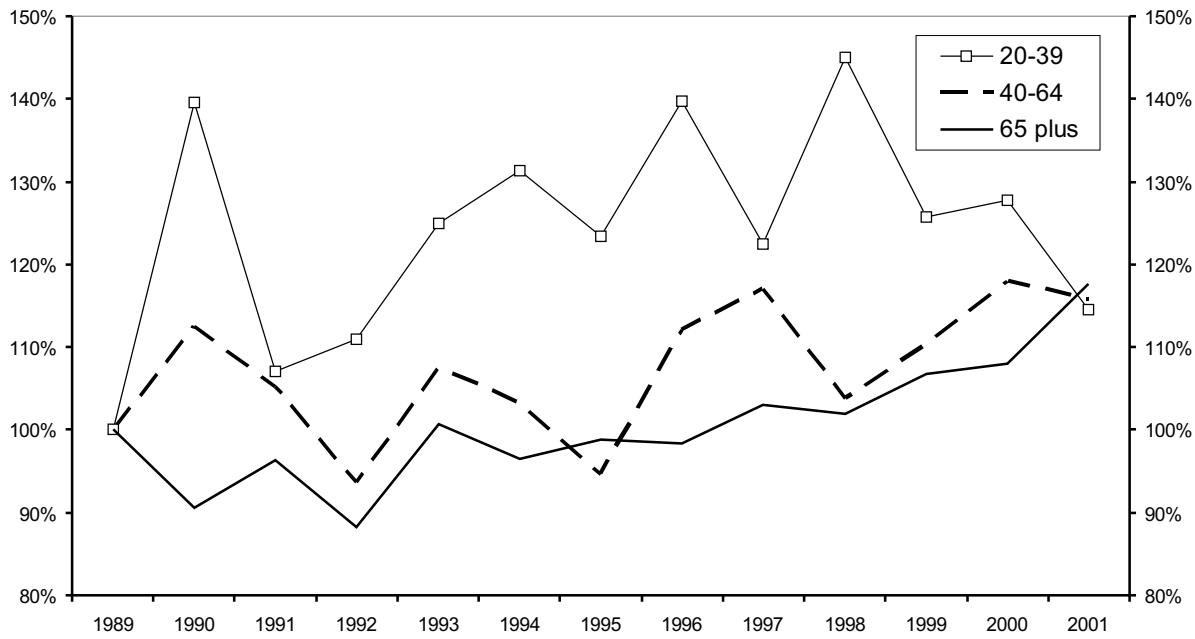
A1.7.2 Pneumonia accounts for around 2% of deaths, for both men and women, up to age 70 but becomes a far more significant cause of death for older ages.

A1.7.3 At first sight, Figures A1.7(M) and A1.7(F) display a rather odd rise and fall in pneumonia mortality over the 1990s. In fact the pneumonia mortality rates have been heavily influenced by two changes in coding practice: first in 1993 following the introduction of automated coding of cause of death by O.N.S. which led to an increase in the number attributed to pneumonia; second the recent decrease due to changes in coding causes of death following the introduction of ICD-10.



Source: own figures based on O.N.S. data (2003)

Figure A1.5b(M). Mortality rates from other accidents 1989 to 2001 as a proportion of the rate in 1989, males in England and Wales by age group

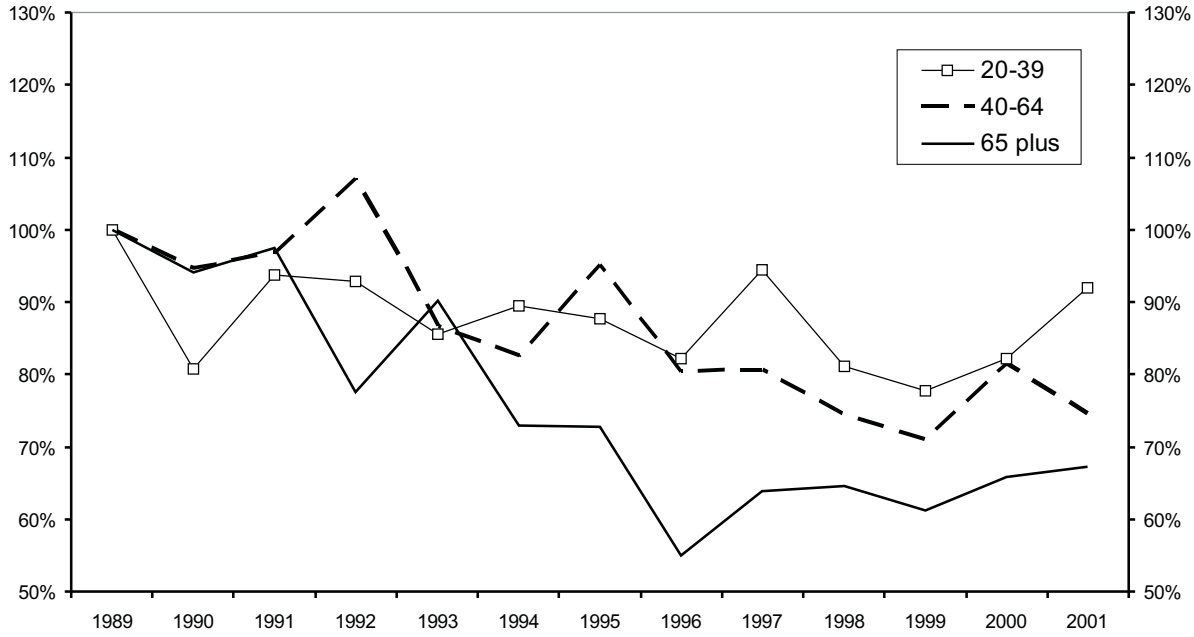


Source: own figures based on O.N.S. data (2003)

Figure A1.5b(F). Mortality rates from other accidents 1989 to 2001 as a proportion of the rate in 1989, females in England and Wales by age group

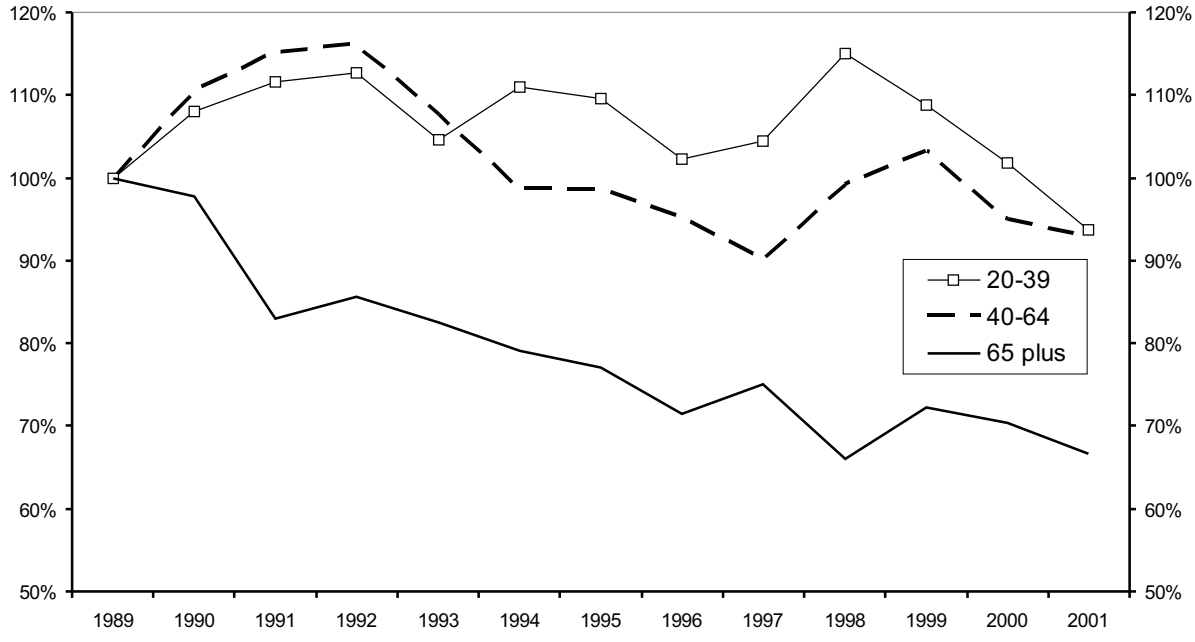
Source: own figures based on O.N.S. data (2003)

Figure A1.5c(M). Mortality rates from violent deaths 1989 to 2001 as a proportion of the rate in 1989, males in England and Wales by age group



Source: own figures based on O.N.S. data (2003)

Figure A1.5c(F). Mortality rates from violent deaths 1989 to 2001 as a proportion of the rate in 1989, females in England and Wales by age group



Source: own figures based on O.N.S. data (2003)

Figure A1.5d(M). Mortality rates from suicide 1989 to 2001 as a proportion of the rate in 1989, males in England and Wales by age group

Source: own figures based on O.N.S. data (2003)

Figure A1.5d(F). Mortality rates from suicide 1989 to 2001 as a proportion of the rate in 1989, females in England and Wales by age group

Table A1.6. Deaths from AIDS, cause-specific mortality rate per 100,000 lives and deaths as a percentage of all deaths, England and Wales population, 2001

	Age group						
	20-29	30-39	40-49	50-59	60-69	70-79	80+
Males							
Deaths per 100,000	0.3	1.1	1.2	0.5	0.3	0.2	0.0
As % of all causes	0%	1%	1%	0%	0%	0%	0%
Females							
Deaths per 100,000	0.3	0.4	0.4	0.0	0.2	0.0	0.0
As % of all causes	1%	1%	0%	0%	0%	0%	0%

Own figures — data source: O.N.S. (2003)

Table A1.7. Deaths from pneumonia, cause-specific mortality rate per 100,000 lives and deaths as a percentage of all deaths, England and Wales population, 2001

	Age group						
	20-29	30-39	40-49	50-59	60-69	70-79	80+
Males							
Deaths per 100,000	1	2	5	11	33	164	1,114
As % of all causes	1%	2%	2%	2%	2%	3%	9%
Females							
Deaths per 100,000	1	1	3	6	22	121	1,069
As % of all causes	2%	2%	2%	2%	2%	4%	9%

Own figures — data source: O.N.S. (2003)

A1.7.4 Adjusting for these distortions in the data, the trend appears fairly flat for the over 65s and perhaps gently increasing for the 40 to 64 age group for both men and women. The fall in rates seen since 1996 for younger men is probably the result of reduced mortality for people who are HIV positive. A significant number of people carrying the HIV virus have been reported as dying from causes other than 'AIDS', pneumonia being a prime example.

A1.8 *Recent Trends in Deaths from Drug and Alcohol Abuse and Chronic Liver Disease*

A1.8.1 Table A1.8 shows the relative importance of drug and alcohol abuse and of chronic liver disease as a cause of death in 2001 for the population of England and Wales.

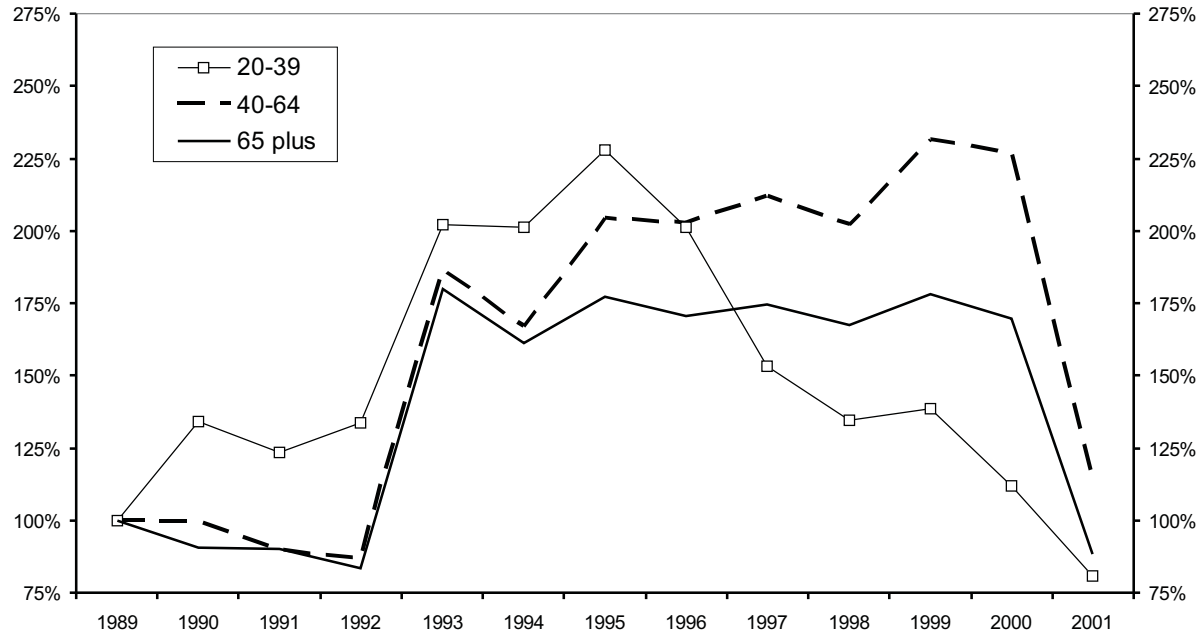
A1.8.2 The number of young adults dying as a result of drug or alcohol abuse is increasing at an alarming pace, as shown in Figures 1.8a(M) and 1.8a(F). In the 11-year period 1989 to 2000, rates for men aged between 20

Source: own figures based on O.N.S. data (2003)

Figure A1.6(M). Mortality rates from AIDS 1989 to 2001 as a proportion of the rate in 1989, males in England and Wales by age group

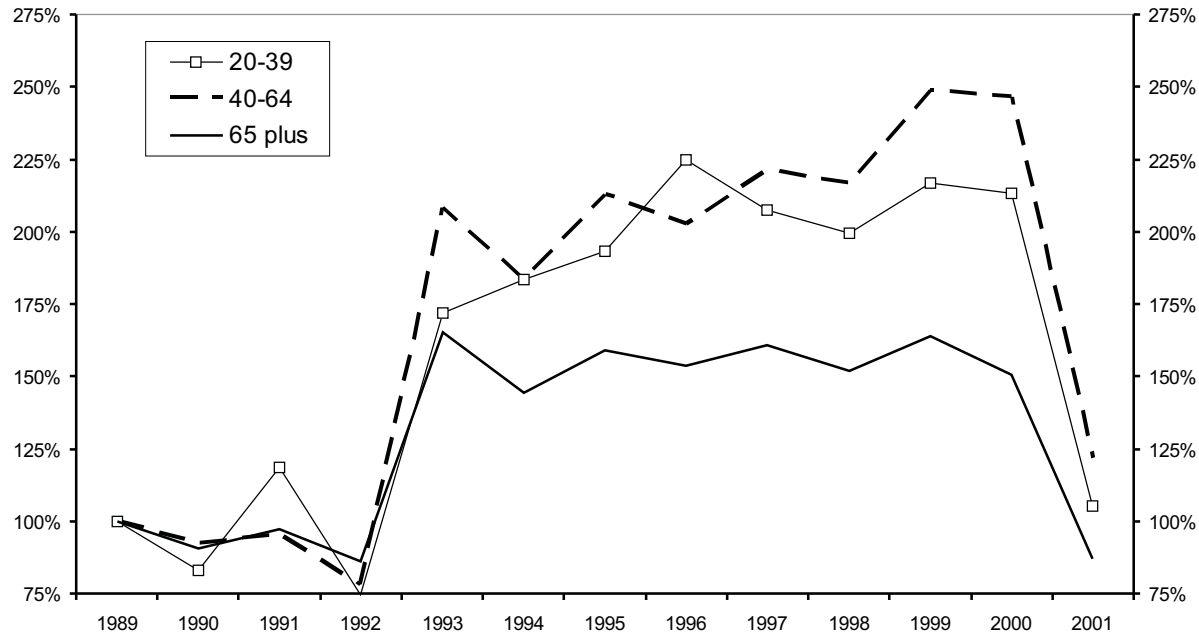
Source: own figures based on O.N.S. data (2003)

Figure A1.6(F). Mortality rates from AIDS 1989 to 2001 as a proportion of the rate in 1989, females in England and Wales by age group



Source: own figures based on O.N.S. data (2003)

Figure A1.7(M). Mortality rates from pneumonia 1989 to 2001 as a proportion of the rate in 1989, males in England and Wales by age group



Source: own figures based on O.N.S. data (2003)

Figure A1.7(F). Mortality rates from pneumonia 1989 to 2001 as a proportion of the rate in 1989, females in England and Wales by age group

Table A1.8. Deaths from drug and alcohol abuse and chronic liver disease, cause-specific mortality rate per 100,000 lives and deaths as a percentage of all deaths, England and Wales population, 2001

	Age group						
	20-29	30-39	40-49	50-59	60-69	70-79	80+
Males							
Deaths per 100,000							
Drugs and alcohol abuse	9	7	5	4	2	1	1
Chronic liver disease	1	7	24	34	32	30	32
As % of all causes							
Drugs and alcohol abuse	11%	6%	2%	1%	0%	0%	0%
Chronic liver disease	1%	6%	10%	6%	2%	1%	0%
Females							
Deaths per 100,000							
Drugs and alcohol abuse	1	1	1	2	1	0	0
Chronic liver disease	0	3	13	17	20	20	18
As % of all causes							
Drugs and alcohol abuse	4%	2%	1%	0%	0%	0%	0%
Chronic liver disease	1%	5%	8%	4%	2%	1%	0%

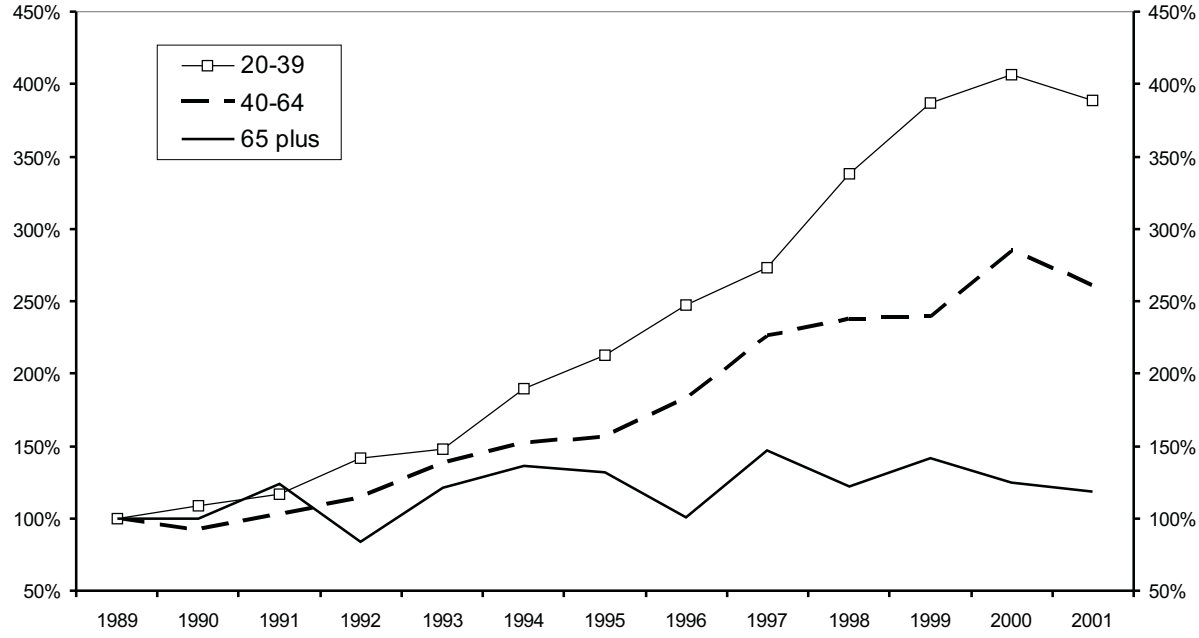
Own figures — data source: O.N.S. (2003)

and 39 increased by over 300%. This cause is becoming an increasingly common reason for premature death. In 2001 it accounted for 11% of all deaths for men in their 20s.

A1.8.3 The extent to which this will impact on the insured population is debatable. However, the use of illicit drugs is widespread among younger adults. In 1996, a survey showed that 29% of men aged between 16 and 29 claimed to have taken illicit drugs in the past year (Department of Health, 1999).

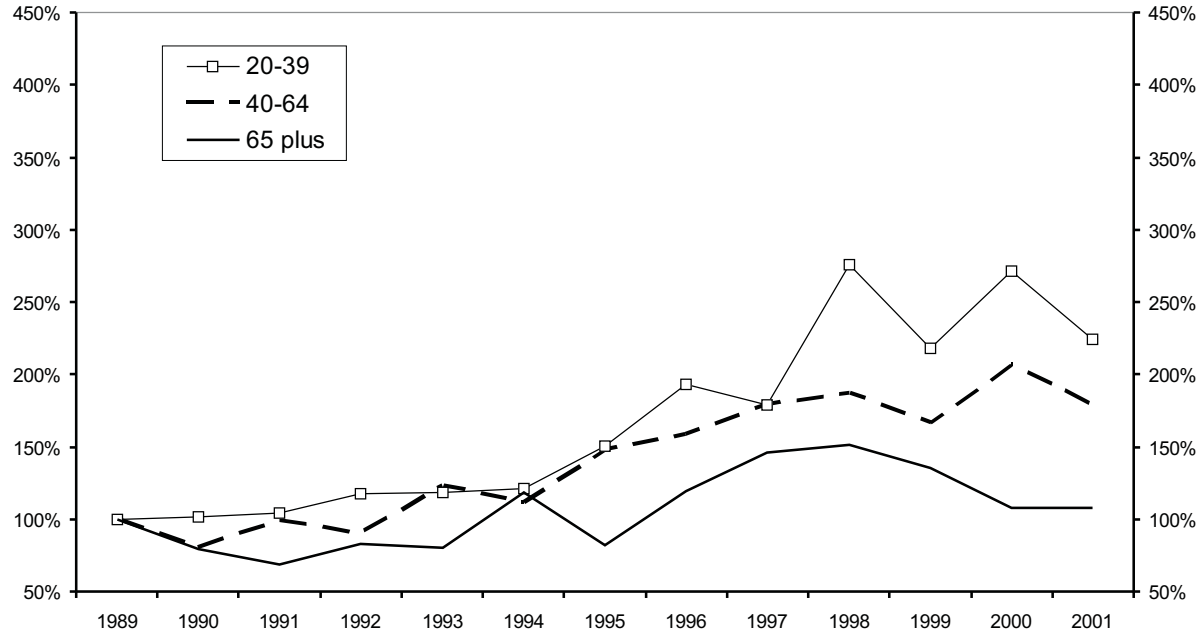
A1.8.4 Changes in liver disease mortality have been similar to those seen for alcohol and drug abuse — see Figures 1.8b(M) and 1.8b(F). It is likely that this is more than coincidental. Hepatitis C — which is common among intravenous drug users — often leads to chronic liver disease. Hepatitis C is discussed further in A2.2.3.

A1.8.5 Chronic liver disease has now become a very significant cause of early death, accounting for 10% of deaths of men in their 40s, and 8% of deaths for women in the same age-group. The adverse trend from deaths due to liver disease contributed minus 0.5% p.a. for men and minus 0.3% p.a. for women to the all cause annual rate of improvement during the 1990s for the 40 to 49 age group.



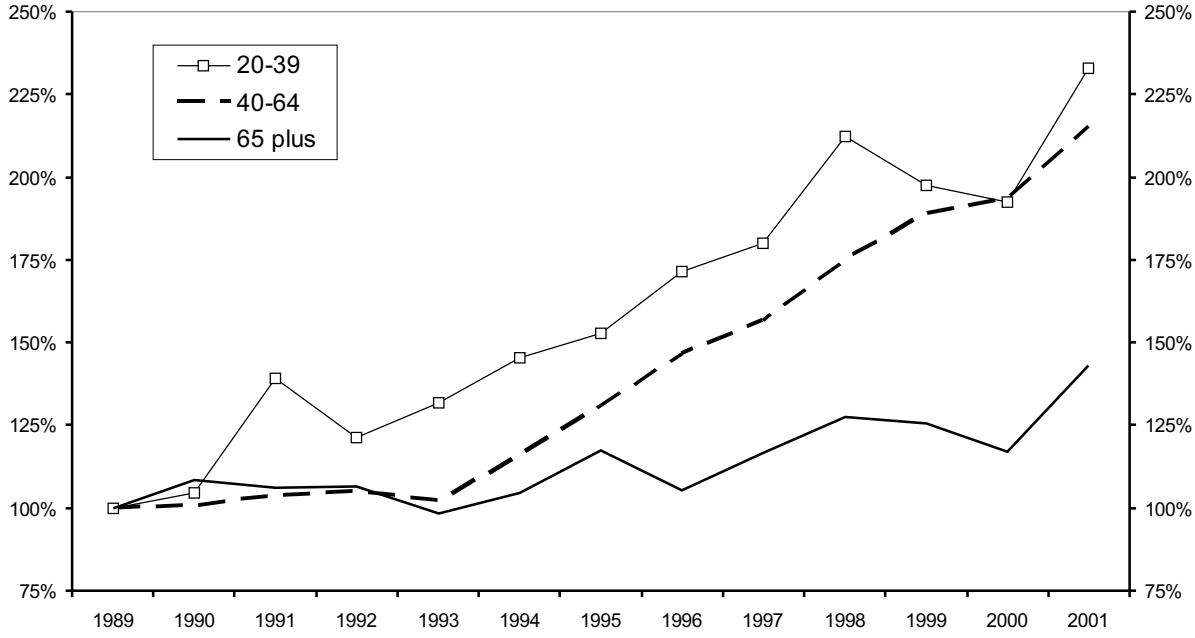
Source: own figures based on O.N.S. data (2003)

Figure A1.8a(M). Mortality rates from drug and alcohol abuse 1989 to 2001 as a proportion of the rate in 1989, males in England and Wales by age group



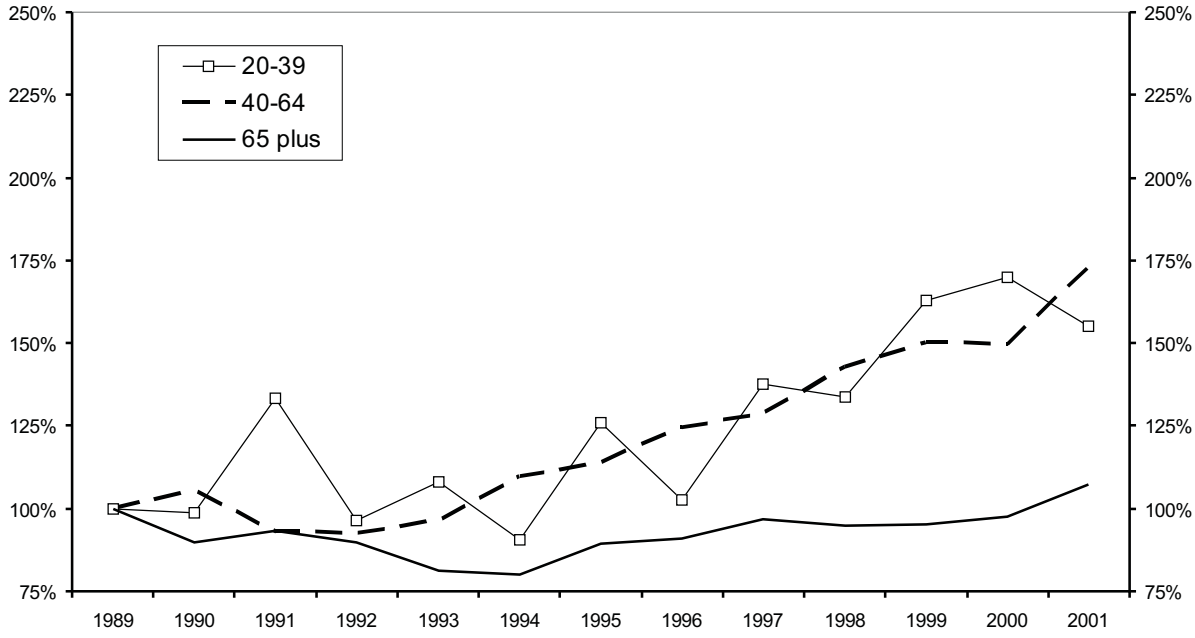
Source: own figures based on O.N.S. data (2003)

Figure A1.8a(F). Mortality rates from drug and alcohol abuse 1989 to 2001 as a proportion of the rate in 1989, females in England and Wales by age group



Source: own figures based on O.N.S. data (2003)

Figure A1.8b(M). Mortality rates from chronic liver disease 1989 to 2001 as a proportion of the rate in 1989, males in England and Wales by age group



Source: own figures based on O.N.S. data (2003)

Figure A1.8b(F). Mortality rates from chronic liver disease 1989 to 2001 as a proportion of the rate in 1989, females in England and Wales by age group

APPENDIX 2

THE THREAT FROM INFECTIOUS DISEASES

A2.1 *Introduction*

A2.1.1 This appendix offers a more detailed review of the threat of infectious diseases on mortality in the U.K. The discussion will be confined to examples of viruses, bacteria and prions that threaten public health in the U.K. and other parts of the world.

A2.1.2 Viruses are the most frequent cause of infectious disease in humans. Examples of life-threatening diseases caused by viruses include influenza and acquired immuno-deficiency syndrome (AIDS). Viruses are the smallest of the infectious agents with the possible exception of prions. Unlike bacteria or other living organisms, viruses do not have any cellular structures. They are single particles that contain genetic material. They cannot replicate on their own but require to enter other cells to help them replicate (Myint *et al.*, 1999). Some viral infections can be prevented by vaccination or treated with anti-viral drugs.

A2.1.3 Bacteria are single-celled micro-organisms that multiply by splitting into two. They are responsible for life-threatening diseases such as tuberculosis, pneumonia and food poisoning. Illnesses caused by bacteria can be treated with antibacterials. However, resistance to antibacterials has been an increasing problem over recent decades (Myint *et al.*, 1999).

A2.1.4 Prions are believed to be abnormal proteins that propagate without any genetic material (Dormont, 2002), unlike viruses or bacteria. They are responsible for fatal human diseases including variant Creutzfeldt-Jakob disease (vCJD) which emerged in the U.K. in the mid 1990s. There is currently no vaccine or treatment for prion-related diseases in humans.

A2.2 *Viral Infections*

A2.2.1 *AIDS in the U.K.*

A2.2.1.1 AIDS is a fatal condition that was first recognised in 1981 and is caused by infection with the human immuno-deficiency virus (HIV). HIV destroys a subset of the body's immune system called the CD4+ cells, making the person 'immunocompromised' and, therefore, vulnerable to a wide range of infection and some forms of cancer. An infected individual is regarded as having AIDS when they develop an 'AIDS defining illness' which includes specified infections or cancers. Without anti-viral treatment, it would take about 3 to 8 years for a newly infected individual to develop AIDS. However, the development of the condition can be considerably delayed by treating patients with highly active antiretroviral therapy

Table A2.2.1.2. HIV infected individuals by gender and their latest reported stage, cumulative data to end of December 2002 in the U.K.

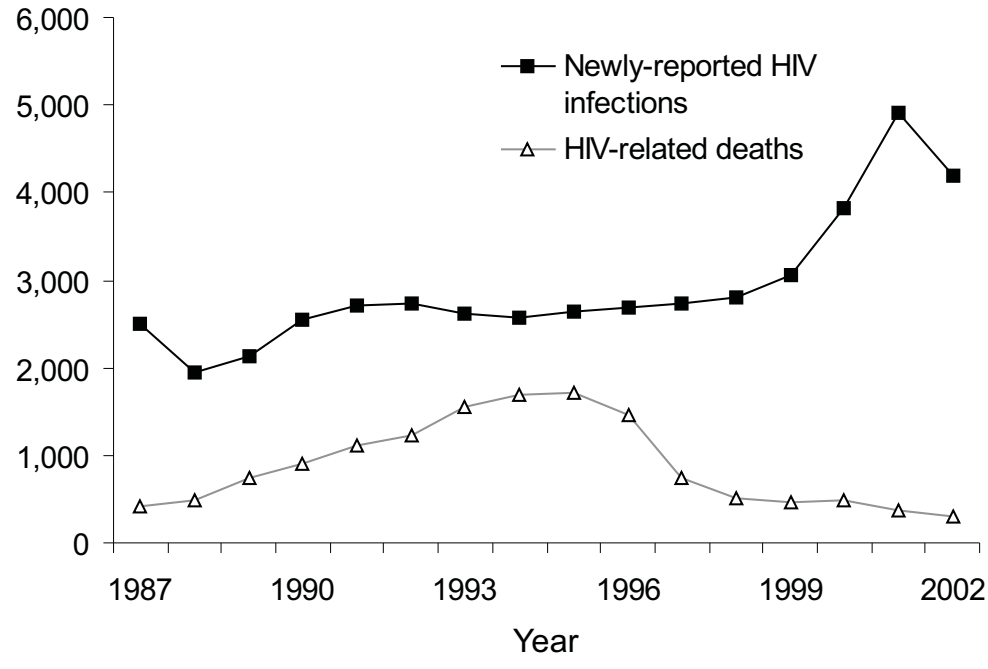
	Male	Female	Total
Infection reported only	23,565	9,035	32,600
AIDS but not death reported	5,123	1,499	6,622
Death with AIDS reported	11,239	1,305	12,544
Death without AIDS reported	2,041	411	2,452
Total (plus 43 with no stated gender)	41,968	12,250	54,261

Source: P.H.L.S., 2003c.

(HAART). A concerning development is the increased detection of drug-resistant strains of HIV (Yerly *et al*., 1999).

A2.2.1.2 The Public Health Laboratory Services (P.H.L.S.) has recorded 54,261 HIV-infected individuals from the beginning of the HIV epidemic in the U.K. in the early 1980s to the end of 2002 (Table A2.2.1.2). Twenty three percent of these individuals have died of AIDS and 5% have died without reported AIDS. P.H.L.S. records show that there were 39,000 individuals living with HIV in the U.K. at the end of 2002, although the actual number including unreported cases is expected to be higher (P.H.L.S., 2003c).

A2.2.1.3 The number of newly-reported cases of HIV infection in the



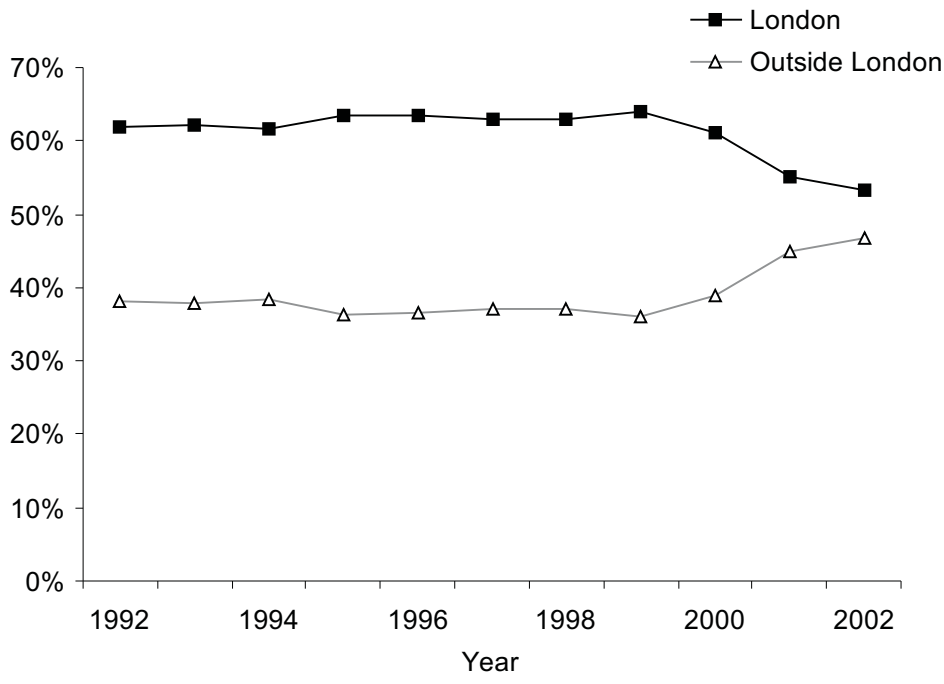
Source: P.H.L.S.

Figure A2.2.1.3. Number of newly-reported HIV infections and HIV-related deaths in the U.K. 1987-2002

U.K. has increased over the period 1987 — 2002 as shown in Figure A2.2.1.3 (P.H.L.S., 2003c). The number rose noticeably from 1999 to 2001. This could be due to a change in P.H.L.S. surveillance policy from January 2000 whereby clinicians were required to report newly-diagnosed cases, in addition to those reported by laboratories. There is also uncertainty regarding the effect of the promotion of HIV testing on the reporting of new HIV infection over the years. Nevertheless, P.H.L.S. data provide reasonable records to investigate the trends of morbidity and mortality of HIV infection in the U.K.

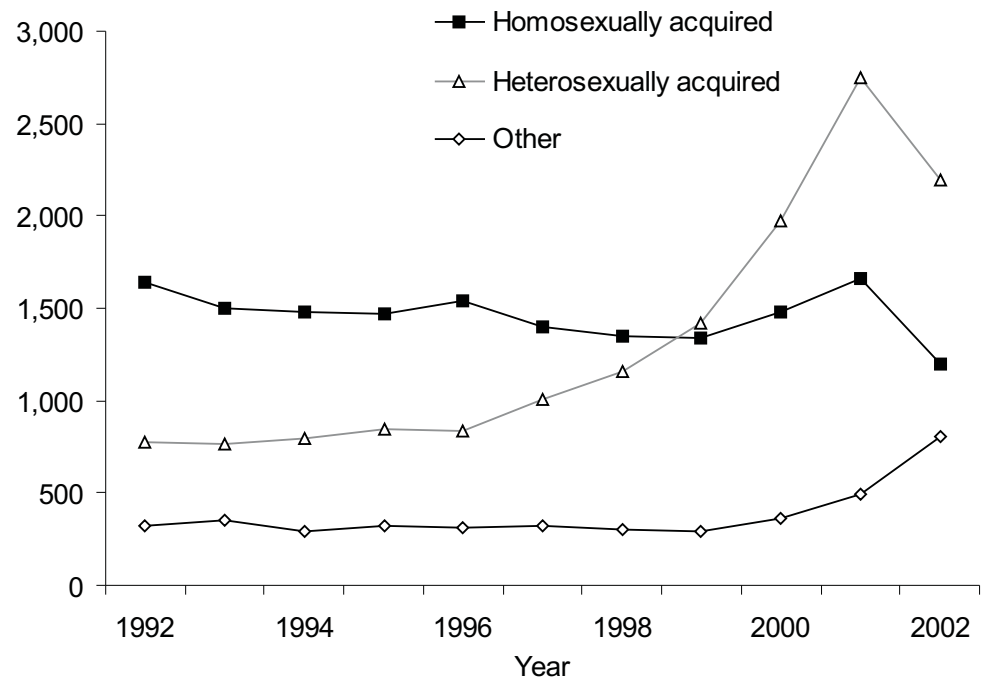
A2.2.1.4 On average 62% of newly-diagnosed HIV cases were reported in London between 1992 and 1999. However, this percentage has gradually reduced from about 64% in 1999 to 53% in 2002 (Figure A2.2.1.4). The proportion of newly-diagnosed HIV cases outside London has increased markedly since 1999.

A2.2.1.5 The U.K. has seen a major shift in the mode of HIV transmission, from homosexually-acquired to heterosexually-acquired infection over the last decade (P.H.L.S., 2003c). For example, 60% of newly-reported infections were attributable to sex between men in 1992. By 2002, this figure had dropped to 28%. The corresponding figures attributable to sex between men and women were 28% in 1992 and 52% in 2002. The actual number of newly-reported HIV cases attributable to sex between men has



Source: P.H.L.S.

Figure A2.2.1.4. Newly-reported HIV-infections in and outside of London as a percentage of total reported infections in the U.K., 1992-2002



Source: P.H.L.S.

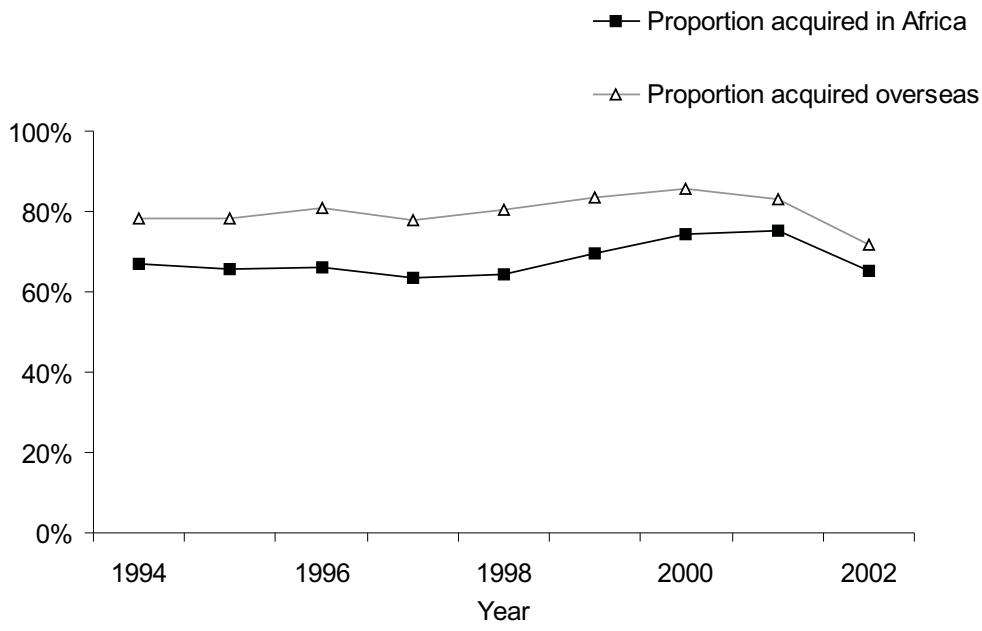
Figure A2.2.1.5. Number of HIV infections in the U.K. by mode of transmission, 1992-2002. Infections classified as 'other' include transmission by intravenous drug use, blood transfusion and transmission from mother to infant

been relatively stable since 1992. However, the number of newly-reported HIV cases due to sex between men and women has escalated rapidly since 1997 and became the major mode of transmission in 1999 (see Figure A2.2.1.5).

A2.2.1.6 The impact of international travel on heterosexual transmission of HIV within the U.K. population cannot be ignored. Between 1994 and 2002, an average of 80% of heterosexually-acquired HIV cases were thought to have become infected overseas. The corresponding figure for those infected in Africa alone is 68% (P.H.L.S., 2003c).

A2.2.1.7 Data for HIV infection reported in 2002 show various patterns regarding ethnicity, gender and mode of infection among the newly-reported HIV individuals (see Table A2.2.1.7). Among infected people who considered themselves 'white', there were more men (84%) than women (16%). This is in contrast to those who considered themselves 'black', where there were more women (61%) than men (39%). The major mode of transmission for 'white' men was sex between men (75%), whilst for 'black' men it was sex between men and women (78%), (P.H.L.S., 2003a). For women of all ethnic backgrounds heterosexual infection was the major mode of transmission (P.H.L.S., 2003a).

A2.2.1.8 There has also been a shift in gender trends with an increasing



Source: P.H.L.S.

Figure A2.2.1.6. Proportion of heterosexually-acquired infections reported in the U.K, that were acquired overseas and proportion that were acquired in Africa, 1994-2002

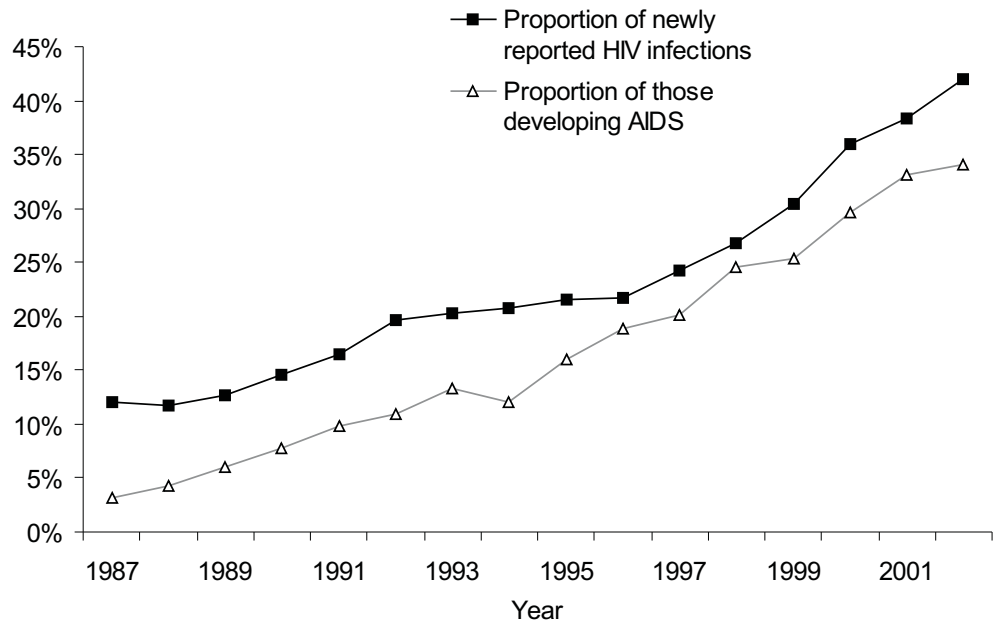
Table A2.2.1.7. Newly-reported HIV-infected individuals in 2002 analysed by gender and ethnicity

	White	Black	Other	Not known	Total
Male	1,228	965	101	790	3,084
Female	240	1,578	66	370	2,254
Total	1,468	2,543	167	1,160	5,338

Source: P.H.L.S., 2003a

number of women reported to be infected with HIV in the U.K. (P.H.L.S., 2003d). For example, only 12% of newly-reported HIV individuals in 1987 were women but this figure rose to 42% in 2002 (Figure A2.2.1.8). The rise in infections in females has been observed globally since the 1980s. In 2002, statistics from WHO has, for the first time, indicated equal numbers of infected men and women (Quinn, 2003).

A2.2.1.9 Most of the HIV-infected women are of childbearing age. In 2001, there were about 561 childbirths by HIV-infected women with an estimated 49 newborns infected with the virus (P.H.L.S., 2003b). With an increasing number of women infected with HIV, and about 37% of women unaware of their positive status, maternal infection could be a potential threat within the U.K. population (P.H.L.S., 2003b). However, maternal



Source: P.H.L.S.

Figure A2.2.1.8. Proportion of those reporting new HIV infections that are female and proportion of those developing AIDS that are female, U.K., 1987-2002

infection may be prevented by diagnosis of HIV infection prior to delivery followed by the use of appropriate interventions such as antiretroviral treatment and prophylaxis (Quinn, 2003; P.H.L.S., 2003b).

A2.2.1.10 Deaths of HIV-infected individuals in the U.K. escalated sharply from the mid 1980s and early 1990s before reaching a peak in the mid 1990s (P.H.L.S., 2003c). Since the mid 1990s, HIV death rates in the U.K., as in other high-income countries, have fallen dramatically (P.H.L.S., 2003c). This fall in the number of deaths is related to the introduction of HAART in 1996.

A2.2.1.11 However, HAART has many serious side effects and the long-term effects on ageing HIV-infected individuals are not yet apparent. The treatment has been reported to increase the risks of heart attack by 27% per year of cumulative HAART use (Lucas, 2003). The use of protease inhibitors in HAART has also been shown to increase cardiovascular diseases by 200%. There is also evidence that HIV infection itself, despite treatment with HAART, increases the risk of heart disease by 63%. Apart from affecting the cardiovascular system, HAART is also toxic to the bone, kidney, liver and fat cells (for a review see Lucas, 2003). These indicate that HAART may reduce the life expectancy of a person receiving the treatment. Nevertheless life expectancy without the treatment would be dramatically lower.

A2.2.2 *The Global AIDS Pandemic*

A2.2.2.1 The Joint United Nations Programme on HIV/AIDS (UNAIDS) and WHO released an annual report on the status of the global AIDS pandemic up to December 2002 (UNAIDS/WHO, December 2002). Some summary statistics from the report are given in Tables A2.2.2.1a and A2.2.2.1b.

The WHO report identified four major trends in the pandemic:

— An increasing number of women infected with HIV globally;
— Threats to economic activities and political stability, especially in Africa;
— Effective efforts to prevent HIV infection in some regions; and
— Lack of resources to continue funding efforts to prevent HIV infection, particularly in Africa.

A2.2.2.2 The number of people living with HIV was estimated at 42 million in December 2002 — about 50% higher than the number projected by WHO in 1991. In 2002, there were about 5 million newly-infected cases and

Table A2.2.2.1a. Global estimates of HIV infection

	Million
Number of people living with HIV/AIDS	42.0
New infections in 2002	5.0
HIV/AIDS death in 2002	3.1

Source: UNSAIDS/WHO, AIDS Epidemic Update, December 2002

Table A2.2.2.1b. Regional HIV statistics

Region	Total number of HIV-infected individuals	Number of new infections in 2002	Estimated deaths from HIV/AIDS during 2002
Sub-Saharan Africa	29,400,000	3,500,000	2,400,000
North Africa and Middle East	550,000	83,000	37,000
South and South-East Asia	6,000,000	700,000	440,000
East Asia and Pacific	1,200,000	270,000	45,000
Latin America	1,500,000	150,000	60,000
Caribbean	440,000	60,000	42,000
Eastern Europe and Central Asia	1,200,000	250,000	25,000
Western Europe	570,000	30,000	8,000
North America	980,000	45,000	15,000
Australia and New Zealand	20,000	500	<100
Total	42,000,000	5,000,000	3,100,000

Source: UNAIDS/WHO, AIDS Epidemic Update, December 2002

half of them were young people between the ages of 15 and 24. HIV killed 3.1 million people globally in 2002 alone (for more data see Tables A2.2.2.1a and A2.2.2.1b). Without effective vaccines and treatment, the HIV pandemic is not expected to abate in the near future.

A2.2.2.3 Sub-Saharan Africa remains the hardest-hit region. In 2002, there were 2.4 million deaths due to AIDS and around 3.5 million new HIV infections. From 1992 to 2002, the estimated number of HIV-infected people almost tripled, rising from about 11 million to 29 million. In some countries, such as Botswana and Zimbabwe, the prevalence of HIV has risen to the worryingly high level of over 30% of the population. In some other regions, however, such as Senegal, the prevalence was as low as under 2% in 2002. The incidence of HIV has been observed to stabilise or decline in some parts of Africa including Uganda, Zambia and Ethiopia. However, limited resources for HIV/AIDS treatment are a continuing problem in southern Africa. Despite growing global awareness of the situation, it is likely that the epidemic will continue to have a devastating impact on the economies of Sub-Saharan African countries.

A2.2.2.4 The second most affected region is Asia and the Pacific where the epidemic has claimed about half a million lives since the late 1980s. At the end of 2002, about 7.2 million people were living with HIV — a 10% increase over the previous year. With China, India and Indonesia making up half of the world's population, epidemics in these countries would contribute much to the statistics of HIV infection. India has a HIV prevalence rate of less than 1%, equaling 4 million people — the highest in the world after South Africa. China currently has 1 million infected individuals and the number is estimated to reach 10 million by the end of the decade, owing to the spread of HIV through intravenous drug use, contaminated blood donations and heterosexual transmission. Uncontrolled epidemics may have a devastating effect on the productivity of these countries, which in turn may affect the global economy.

A2.2.2.5 Latin America and the Caribbean have the third highest number of people living with HIV. An estimated 1.9 million people were living with HIV at the end of 2002, of whom 210,000 were newly-infected. Homosexual and heterosexual transmissions have been the main modes of transmission. Infection through the sharing of drug-injecting equipment has also been increasingly prominent. State-sponsored treatment has been made available in several countries such as Brazil, Argentina and Cuba. The financial strain caused by AIDS may add a further burden to the existing financial problems of some of these countries.

A2.2.2.6 The fastest growing HIV epidemic regions are Eastern Europe and Central Asia. In 2002, there were about 250,000 new infections. Some countries like Georgia and Uzbekistan have experienced explosive growth. In the first six months of 2002, there were as many new HIV infections as had been reported over the previous ten years.

A2.2.2.7 In high-income countries, the introduction of HAART since 1996 has markedly reduced HIV-related mortality. During 2002, there were about 500,000 receiving HAART among the 1.6 million infected individuals. There has been a continued shift of the epidemic into marginalised populations that lack access to the information and services that could help HIV prevention. Declining mortality due to AIDS has resulted in increasing complacency and reduced prevention efforts.

A2.2.2.8 In summary, HIV remains a threat to global morbidity and mortality as well as the economy. Africa continues to fight the onslaught of AIDS with limited resources, although some improvements have been observed in several countries. Eastern Europe and parts of Asia have become homes to the fastest growing HIV epidemic in the world. In high-income nations, the use of HAART has postponed HIV-related death but the number of infected individuals continues to rise.

A2.2.3 *The Hepatitis C Virus and Mortality in the U.K.*

A2.2.3.1 Mortality rates from liver disease have been on the rise in the U.K. over the past decade as shown in Section 2 (see also Willets, 1999; Deuffic *et al.*, 1999). This rise in mortality has been at least partly attributed to infection with the hepatitis C virus (HCV). The HCV was identified as recently as 1989 but up to 3% of the world population is infected with the virus. HCV has been categorised into six strains called genotypes 1 to 6. Genotype 1 constitutes about 74% of HCV infection in developed countries (Franciscus, 2003).

A2.2.3.2 About 80% of infected individuals are unable to eradicate the virus without treatment and hence become chronic HCV carriers. An estimated 20% of male HCV carriers will develop liver cirrhosis. Other fatal outcomes of HCV infection include chronic active hepatitis and liver cancer, although liver transplant may prolong life substantially (see Bird *et al.*, 2001). HCV-infected individuals are 23.6 times more likely to develop liver cancer than non-infected individuals (see La Vecchia *et al.*, 2000). The incubation periods from HCV infection to cirrhosis and to liver cancer are long and measured in decades (Bird *et al.*, 2001).

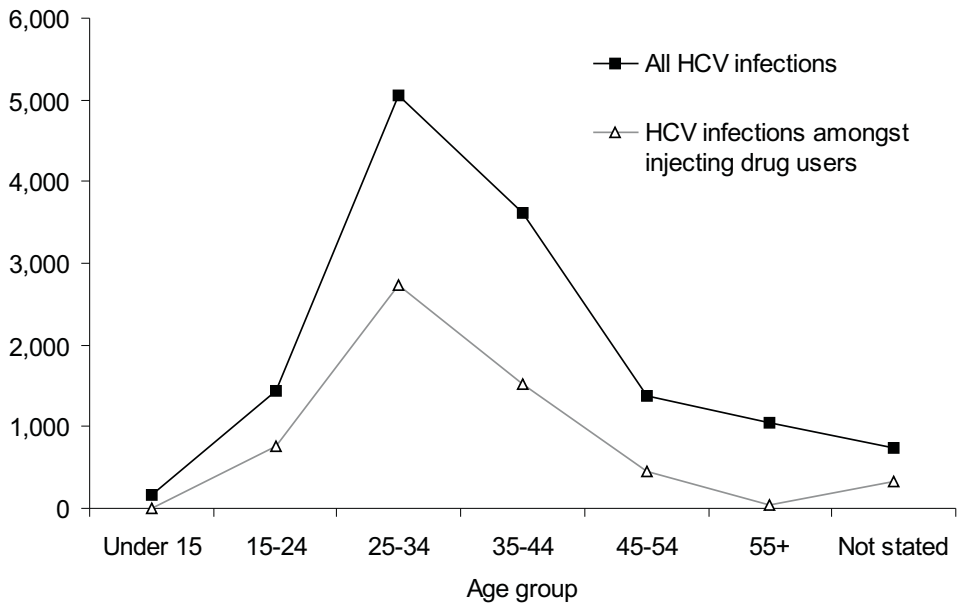
A2.2.3.3 About 0.5% of the U.K. population may be chronic HCV carriers (see Deuffic *et al.*, 1999). This suggests that about 300,000 individuals are infected, making the scale of HCV epidemic several times larger than that of HIV in the U.K. An epidemic of HCV is thought to have started after World War II mainly through blood transfusion, and has been further amplified by needle sharing among intravenous drug users (Bird *et al.*, 2001; Deuffic *et al.*, 1999). Since 1991, all U.K. blood donations have been screened for HCV antibodies. The prevalence of HCV amongst intravenous drug users has been estimated at 76% in Scotland and 49% in England and Wales (Bird *et al.*, 2001). The majority of HCV carriers in the U.K. are under the age of 45. Bird *et al.* (2001) reported that the modal age

group at HCV diagnosis was 25-34 according to data up to 1998. HCV-infected injectors tend to be younger (see Figures A2.2.3.3a and A2.2.3.3b).

A2.2.3.4 There is currently no vaccine to prevent the type of HCV infection which is related to 70% of all liver cancers in developed countries (see La Vecchia *et al.*, 2001). Eradication of the virus would, therefore, be expected to reduce the incidence of liver cancers and other fatal liver disease, hence reducing mortality attributable to these problems.

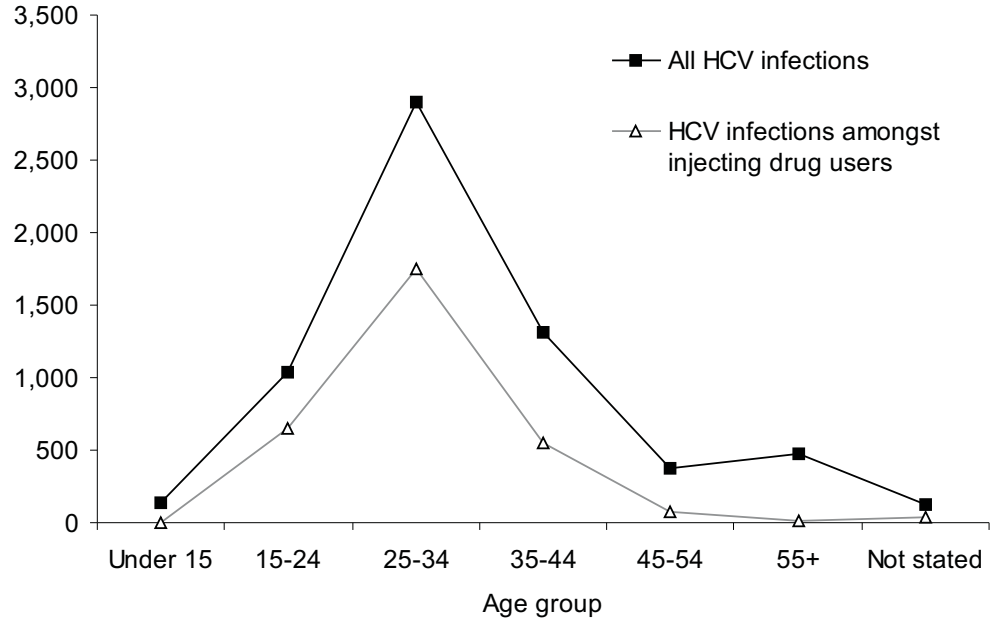
A2.2.3.5 Treatment of chronic HCV infection is currently based on a combination of pegylated interferon-alpha and ribavirin. The treatment can lead to clearance in 50% of genotype 1 infection and 80% of genotype 2/3 infection (Pawlotsky, 2003). Success rates of the current HCV treatment are dependent on viral loads, with lower viral loads leading to a greater success rate. The effectiveness of the treatment may also be affected by the method of manufacturing pegylated interferon-alpha (Franciscus, 2003).

A2.2.3.6 The eradication rates of the current treatment are higher than the 20% eradication rate reviewed by Willets in 1999, indicating an improvement in HCV treatment. It remains to be seen if this advancement in treatment, together with effective blood screening to prevent HCV transmission, might eventually lead to a fall in mortality rates from HCV-related liver diseases in the U.K.



Source: Bird *et al.*, 2001

Figure A2.2.3.3a. Number of reported and confirmed HCV infections in England and Wales by age group to end December 1998. Note: the actual number of HCV-infected individuals is thought to be higher than these recorded cases



Source: Bird *et al.*, 2001

Figure A2.2.3.3b. Number of reported and confirmed HCV infections in Scotland by age group to end December 1997. Note: the actual number of HCV-infected individuals is thought to be higher than these recorded cases

A2.2.4 *Influenza*

A2.2.4.1 Influenza is related to at least 3,000 to 4,000 deaths per year in the U.K. Death rates from pneumonia and influenza are much higher among people above the age of 65 (some 797 deaths per 100,000 healthy people) compared with those under 65 (2 deaths per 100,000) (Liddle & Jennings, 2001).

A2.2.4.2 During an epidemic in 1989-90, over 20,000 deaths were reported in the U.K. In the 1918-19 'Spanish Flu' pandemic, influenza is estimated to have killed 20 to 40 million people world-wide (Liddle & Jennings, 2001). In the 20th century, there were four pandemics which may all have originated in parts of southern China. In this region some people live in close proximity to animals such as ducks (the breeding ground for new strains) and pigs (the suspected intermediary for transmission to humans). This increases the chances of transmission of new strains of influenza virus from animals to humans. Increasing air travel can help spread the new strains to other parts of the world.

A2.2.4.3 There are three types of influenza virus called influenza A, B and C which are characterised by some proteins inside the virus. These are further classified according to glycol-proteins called haemagglutinin (H) and neuraminidase (N) on the surface of the viruses. Each year, the WHO

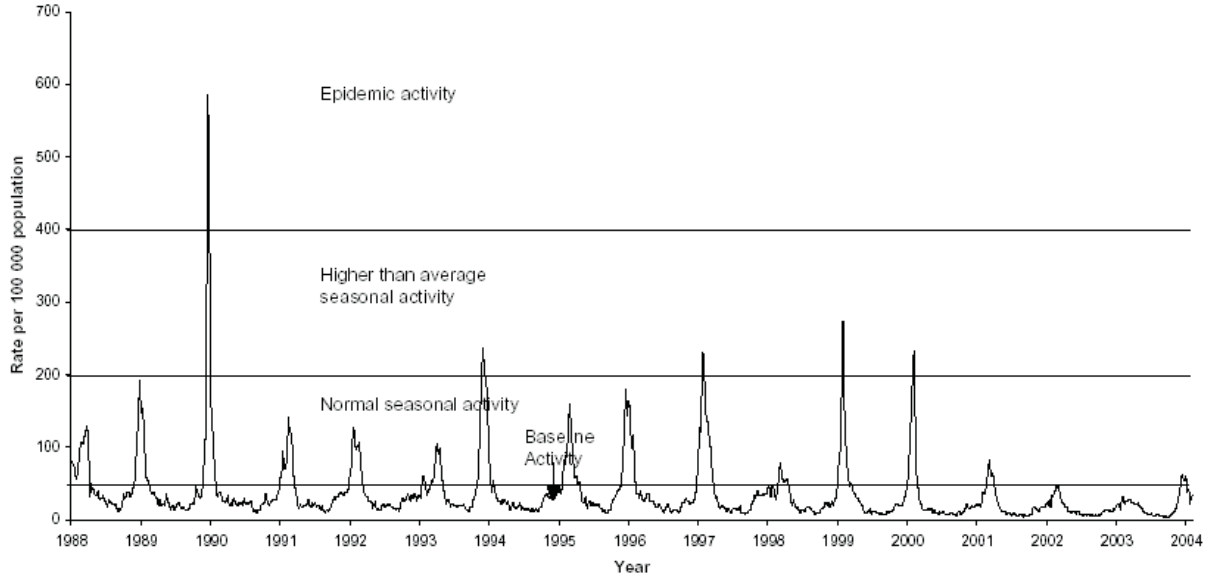
recommends the appropriate vaccine against the expected influenza strains that may cause epidemics, according to global surveillance data.

A2.2.4.4 Vaccine against influenza is safe in the elderly. It is also cost-effective according to studies conducted in the U.S.A. (for a review see Liddle & Jennings, 2001). Major reductions in morbidity and mortality have been observed in North America where the vaccine has been used relatively extensively. In the U.K., National Health Service policy sets a target of 70% uptake in people over 65 years old and laboratory reports of infections attributable to influenza have reduced since 1999-2000 in the U.K. (see Figure A2.2.4.4). Whether this is due to effective implementation of immunisation programmes or a lack of influenza activity remains uncertain.

A2.2.4.5 Although vaccine and drugs against influenza are available, they may not be effective against new strains of influenza. The emergence of new strains of influenza may be able to inflict large numbers of deaths as demonstrated by previous epidemics and pandemics. As deaths from cancer and heart disease reduce in the U.K., deaths from infectious diseases including influenza could become an increasingly significant factor in determining old-age mortality.

A2.2.5 *Severe Acute Respiratory Syndrome (SARS)*

A2.2.5.1 SARS attracted much media attention world-wide in 2003. It



Source: P.H.L.S.

Figure A2.2.4.4. Laboratory reports of infections due to influenza by date of report for the U.K.

emerged in Guangdong Province, China, where several hundreds of atypical, severe pneumonia cases were reported by late 2002. On 21 February 2003, an infected medical doctor who had treated SARS patients carried the virus out of Guangdong Province into a hotel in Hong Kong. Several infected guests in the hotel then brought the virus by air travel to Vietnam, Singapore and Canada during February and March 2003. The disease continued to spread globally. By June 2003, there had been 790 deaths and 8,445 reported cases in 32 countries, despite intensive international efforts to contain the disease. In the U.K., there have been 4 cases of SARS with no deaths reported.

A2.2.5.2 WHO regards SARS a serious threat. A patient with SARS would typically go through an incubation period of 2-7 days showing no symptom of illness, followed by fever, dry cough and shortness of breath (Rota *et al.*, 2003). The incubation period would allow an infected person to travel by air between two cities and spread the disease. The initial symptoms are non-specific and common, making it possible for infected people to escape diagnosis and infect others. Furthermore, death as a result of respiratory failure happens in about 15% of cases. For a person over the age of 65, the mortality rate can exceed 50% (WHO, 2003). Strong evidence now shows that SARS is caused by a new coronavirus called the SARS-CoV (Rota *et al.*, 2003). Coronaviruses have been known for their frequent mutation, hence raising concerns over the future evolution of the outbreak and prospects of vaccine development (Marra *et al.*, 2003). There is currently no vaccine or anti-viral for SARS. This forces health-care workers to resort to very basic measures such as isolation and quarantine of infected people.

A2.3 *Bacterial Infections and Resistance to Antibacterial Drugs*

A2.3.1 *Overview*

A2.3.1.1 Antibacterials, such as penicillin, are agents which can selectively kill or inhibit the growth of bacteria. The term 'resistance to antibacterial' may refer to the intrinsic or acquired ability of a bacterium to avoid being killed or inhibited by an antibacterial to which it is normally susceptible. Acquired antibacterial resistance is a result of exposure to an antibacterial, leading to preferential survival of resistant strains and selective inhibition of susceptible strains. It is, therefore, a consequence of the use of antibacterials in medicine.

A2.3.1.2 However, resistance is more likely to occur when antibacterials are used excessively, such as where they are made available without prescription or used for a longer duration than needed. Resistance may also develop when antibacterials are used as growth supplements in agriculture (Myint *et al.*, 1999; Heath & Breathnach, 2002). Some examples of antibacterial resistance will be described here.

A2.3.2 *Tuberculosis*

A2.3.2.1 Tuberculosis (TB) kills about 2 to 3 million people world-wide annually and is caused by the bacterium *Mycobacterium tuberculosis*. About one third of the world's population (2 billion people) is infected with this bacterium. TB is usually curable with antibacterials but co-infection with HIV and antibacterial resistant strains have further fuelled the current pandemic of TB (P.H.L.S., 2003e).

A2.3.2.2 In the U.K., TB was the major cause of death at the turn of the century. In 1950, there were about 50,000 cases each year in England and Wales. This figure fell to about 5,000 cases in the mid-1980s. The decline was accelerated by the introduction of the BCG vaccine and antibacterials such as streptomycin. Since the mid-1980s, the number of cases has been increasing and currently there are about 7,000 cases each year (P.H.L.S., 2003e).

A2.3.2.3 In recent years, most of the cases have been reported in major cities, particularly London. The worst-affected groups of the population are the homeless and people arriving from countries where TB or HIV is endemic. In 2000, London accounted for 42% of the total reported TB cases in England and Wales. 80% of those infected in London were born overseas. In England and Wales, the proportion of foreign-born TB cases increased from 45% in 1988 to 59% in 1999 and 63% in 2000 (P.H.L.S., 2003e).

A2.3.2.4 About 6% of *Mycobacterium tuberculosis* samples in England and Wales tested in 2000 were resistant to the antibacterial isoniazid, and 1% were 'multiple drug resistant' defined as resistant to at least isoniazid and rifampicin (P.H.L.S., 2003). Globally, resistance to at least one anti-TB drug among new cases ranged from 2% in Uruguay to 37% in Estonia in 2000 (WHO, 2000). Multiple drug resistance ranged from 0% in several countries to 14% in Estonia (WHO, 2000). There has been concern that increasing international travel may help spread various resistant strains into the U.K.

A2.3.3 *Methicillin-resistant Staphylococcus Aureus*

A2.3.3.1 *Staphylococcus aureus* is a bacterium found in the noses of 20-30% of normal healthy individuals and is also found on human skin. Strains of *Staphylococcus aureus* that are resistant to an antibiotic called methicillin are called methicillin-resistant *Staphylococcus aureus* or MRSA. MRSA is also resistant to many other commonly prescribed antibacterials. The bacterium is usually confined to hospitals and rarely threatens the general public. MRSA could be responsible for wound, skin or urinary tract infection. It could also cause pneumonia and 'blood poisoning' among patients in hospitals.

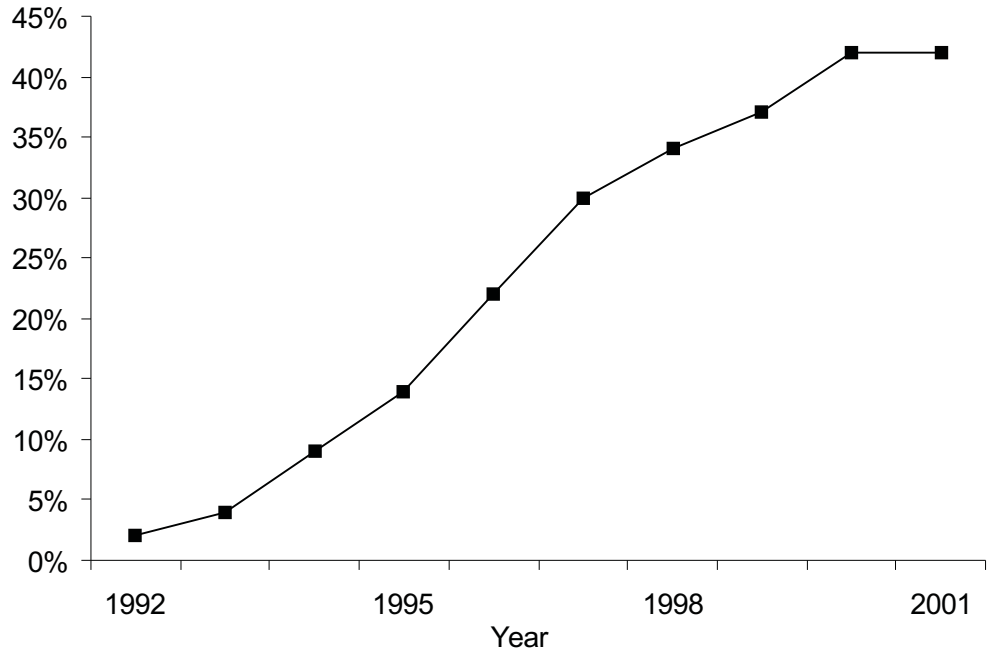
A2.3.3.2 In England and Wales, *Staphylococcus aureus* is the most common cause of 'bacteraemia' defined as the detection of viable bacteria in the blood (P.H.L.S., 2001). The proportion of MRSA in Staphylococcus

aureus bacteraemia samples reported by laboratories increased sharply from 2% in 1992 to 42% in 2001 (see Figure A2.3.3.2a). The number of deaths due to MRSA in the U.K. is thought to have increased over the period 1993-98 as shown in Figure A2.3.3.2b (Crowcroft & Catchpole, 2002; Spencer, 2002).

A2.3.4 *Pneumonia*

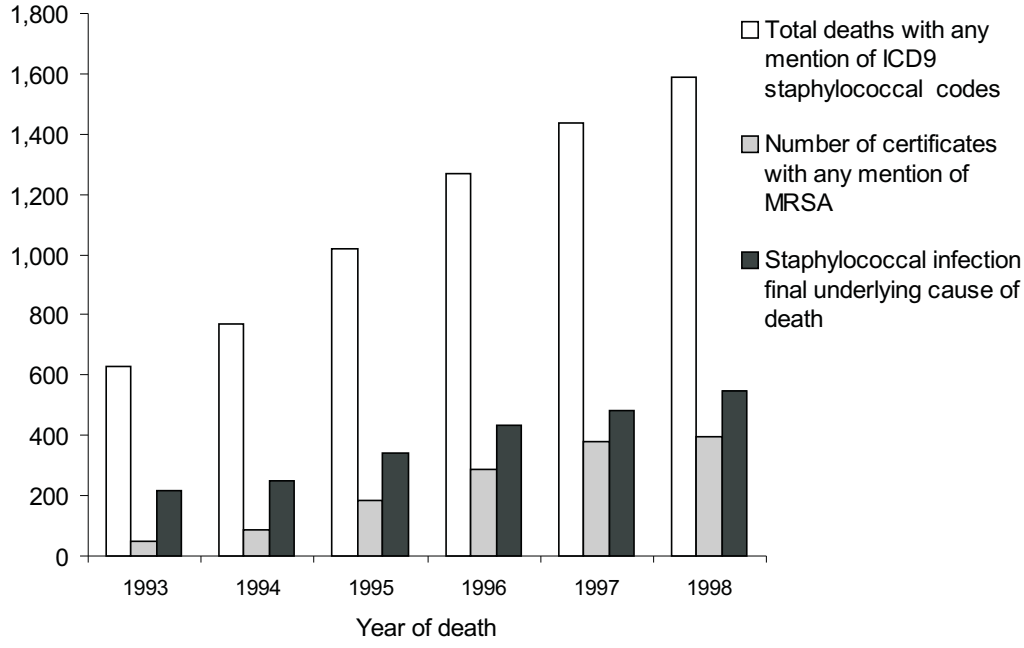
A2.3.4.1 The bacterium *Streptococcus pneumonia* is the most common causative agent of pneumonia — a major cause of death in the U.K. which is responsible for about 10% of deaths among those above the age of 80 (see Sections 2.5 and 2.6). *Streptococcus pneumonia* can also be responsible for other fatal diseases including septicaemia (blood poisoning) and bacterial meningitis. *Streptococcus pneumonia* was the third most common cause of bacteraemia in England and Wales in 2000, after *Staphylococcus aureus* and *Escherichia coli* (*E. coli* O157) (P.H.L.S., 2001).

A2.3.4.2 Resistance of *Streptococcus pneumonia* to penicillin increased from 1% in 1990 to 4% in 1998 and 7% in 2000. Resistance to erythromycin rose from 5% in 1990 to 11% in the mid 1990s and to 22% in 2000 (P.H.L.S., 2001). Many other countries in southern Europe, South-East Asia and USA report much higher rates of resistance to penicillin or erythromycin (P.H.L.S., 2001).



Source: P.H.L.S.

Figure A2.3.3.2a. Proportion of *Staphylococcus aureus* bacteraemia samples that were methicillin-resistant, England and Wales, 1992-2001



Source: Crowcroft & Catchpole, 2002

Figure A2.3.3.2b. Number of death registrations in the U.K with strapphylococcal codes by year of death, 1993-98

A2.3.5 *Food Poisoning*

A2.3.5.1 Cases of food poisoning in the U.K. rose six-fold between 1982 and 2001. In 2001, the number of reported cases was about 1 million and the number of total estimated cases, including unreported ones, was 4.5 million (F.S.A., 2002). The PHLS recorded 65,209 laboratory-confirmed cases of food poisoning caused by the top 5 food-borne bacteria (*Salmonella, E. coli* O157*, Campylobacter, Listeria, and Clostridium Perfringens*) in the U.K. in 2000 (F.S.A., 2002).

A2.3.5.2 *E. coli* O157 is responsible for less than 10% of total food poisoning cases in the U.K.. However, unlike the other causative agents of food poisoning, this bacterium can cause life-threatening diseases such as haemolytic uraemic syndrome (HUS) and thrombotic thrombocytopaenic pupura (TTP) in 2% to 15% of infected individuals (Todd & Dundas, 2001). Mortality rates from HUS are high (3-17%), especially among the elderly (87%). Furthermore, HUS is the most common end-stage renal disease among children in the U.K. (Todd & Dundas, 2001).

A2.3.5.3 The primary reservoir of *E. coli* O157 is cattle. The bacterium can be contracted through contact with infected animals and consuming contaminated food or drinks (Todd & Dundas, 2001). In 2000, resistance of *E. coli* O157 to antibacterials such as streptomycin, suphonamides or tetracyclines was reported at about 14% (P.H.L.S., 2001).

This might reflect the usage of these drugs in cattle. An increasing proportion of multiple drug resistant *E. coli* O157 will be a concern to the health of the nation.

A2.3.5.4 In 2000, the two most commonly reported Salmonella were *S. Thyphimurium* (18%) and *S. Enteritidis* (57%). The proportion of resistance of *S. Thyphimurium* to streptomycin, suphonamides or tetracyclines was above 65%, whilst that of *S. Enteritidis* was under 4%. This may be due to the association of *S. Thyphimurium* with cattle which have been commonly treated with the antibacterials. For *Campylobacter*, another common pathogen in the gut, the proportion of strains resistant to at least one drug was 50% in 2000 (P.H.L.S., 2001). The relatively high proportion of resistance in some of these bacteria is a threat to the health of the U.K. population.

A2.4 *Variant Creutzfeldt-Jakob Disease (vCJD): an example of a prion-related infectious disease*

A2.4.1 The vCJD is a fatal brain disease which emerged in the U.K. during the mid 1990s, following an epidemic of bovine spongiform encephalitis (BSE) or 'mad cow disease' in cattle. The vCJD is thought to have resulted from consuming BSE-infected food. Up to 2 million BSE-infected cattle may have entered the U.K. food chain before a ban on specific bovine parts in 1989 (see Boëlle *et al.*, 2003). Earlier mathematical models predicted possible large numbers of vCJD infection, totalling up to millions in some scenarios (see Willets, 1999). Recent papers have indicated a more optimistic view on the vCJD epidemic, predicting the expected number of infected individuals to be several hundreds but not more than several thousands.

A2.4.2 As of 4 July 2003, 137 cases of vCJD have been diagnosed in the U.K. and of these, 132 have died (U.K. CJD surveillence unit, 2003). National statistics suggest that the vCJD epidemic peaked between 1999 (15 cases) and 2000 (28 cases), (U.K. CJD surveillence unit, 2003). However, there is continued uncertainty over how many people have been infected and how many will eventually die of the condition (Huillard d'Aignaux, 2003). This is mainly due to uncertainties over the exact route of transmission of the disease and the relatively long incubation period. Large scale studies on operated tonsils and appendices are currently being carried out to ascertain the prevalence of vCJD infection in the population. While waiting for more medical data to be made available, mathematical models could serve to estimate the scale of the vCJD problem.

A2.4.3 Using a novel statistical model and the U.K.'s 113 vCJD cases with onset up to December 2000, Boëlle *et al.* (2003) attempted to estimate the incubation period and the extent of the vCJD epidemic. The authors estimated the mean incubation period at 16.4 years (with log-normal distribution), 15.9 years (Gamma distribution) and 14.1 years (Weilbull distribution). The model predicted the size of the epidemic to be between

183 and 304 (Boëlle *et al.*, 2003). This model is dependent on several assumptions such as:

— human exposure to BSE-contaminated food;
— exponentially decreasing susceptibility after 15 years of age; and
— statistical distributions for incubation period.

A2.4.4 Huillard d'Augnaux *et al.* (2003) presented an analysis of the vCJD epidemic to estimate the number of infected persons and to explore the likely future incidence of the disease in the U.K.. The model indicated that the expected numbers of death from vCJD up to the year 2020 are unlikely to exceed 100 cases per year. The authors estimated that the number of infected individuals are of the order of a few hundreds but are unlikely to be more than a few thousands. However, the analysis implied that under some extreme scenarios the number of infected cases could reach millions.

A2.4.5 These predictions are consistent with the work of Ghani *et al.* (2003) which models the transmission of vCJD due to exposure to BSE-infected meat product using 113 cases of vCJD with onset up to the end of December 2000. The authors estimated that the 95% confidence interval for future cases after 2002 to be from 10 to 7,000 deaths. In the short term, Ghani *et al.* (2003) showed that the number of new cases in the 2 to 5 years after 2002 would be relatively low (95% confidence interval for 2 years: 10-80 cases, for 5 years: 10-200 cases). The model also demonstrated that those aged between 10 and 20 years are at the highest risk of infection.

A2.4.6 Taken together, recent papers on mathematical modelling of the vCJD epidemic predict the scale of the epidemic to be of the order of several hundred deaths. Due to uncertainties over the incubation period and mechanism of transmission, the total number of deaths is still unpredictable but unlikely to exceed several thousands. Thus, the vCJD epidemic is not expected to reverse the current trend of improvements in mortality in the U.K.. The vCJD epidemic demonstrates that changes in food production could expose the population to new infectious diseases. With increasing industrialisation of food production in the U.K., problems with a single producer or method of production may affect a large number of people.

A2.5 *The Emergence of New Diseases*

A2.5.1 The emergence of SARS clearly demonstrates that new infectious diseases can still pose a threat to humanity, especially with increasing global air travel. However, experience from SARS also shows that effective international communication and efforts can result in rapid detection and isolation of SARS cases, hence preventing further transmission of the virus. Since the WHO issued a global alert on SARS on 15 March 2003, there has been unprecedented international co-operation involving government agencies, clinicians, epidemiologists and scientists to control the spread of SARS. This readiness in coping with SARS is a result of ongoing international

collaborations to prevent a repeat of global health problems such as the influenza pandemic that killed 20 million people in 1918. Measures set in place to counter bioterrorism have also played a role in containing the virus. Advancement in medical sciences, telecommunication and international networking will continue to help limit potentially devastating effects of new outbreaks like SARS.

A2.5.2 Many infectious diseases have emerged over the last 30 years (see Table A2.5.2), indicating that the danger of new diseases cannot be ignored. Fortunately, most of the new fatal diseases (with the notable exception of AIDS) have limited ability to spread, hence preventing them from becoming major threats to global health (WHO, 2003).

Table A2.5.2. Examples of newly-recognised infectious agents since 1973; adapted from WHO Fact sheet 97, August 1998. www.who.int

Year	Microbe	Disease
1973	Rotavirus	Major cause of infantile diarrhoea globally
1976	Cryptosporidium parvum	Acute and chronic diarrhoea
1977	Ebola virus	Ebola haemorrhagic fever
1977	Legionella pneumophilia	Legionnaires disease
1977	Hantaan virus	Haemorrhagic fever with renal syndrome
1977	Campylobacter jejuni	Enteric diseases distributed globally
1980	Human T-lymphotropic virus 1 (HTLV-1)	T-cell lymphoma-leukemia
1981	Toxin producing strains of *Staphylococcus aureus*	Toxic shock syndrome
1982	*Escherichia coli* O157:H7	Haemorrhagic colitis; haemolytic uraemic syndrome
1982	HTLV-II	Hairy cell leukaemia
1982	Borrelia burgdorferi	Lyme disease
1983	HIV	AIDS
1983	Helicobacter pylori	Peptic ulcer disease
1988	Hepatitis E	Enterically transmitted hepatitis
1990	Guanarito virus	Venezuelan haemorrhagic fever
1991	Encephalitozzon hellem	Conjunctivitis, disseminated disease
1992	*Vibrio cholerae* O139	New strain of epidemic cholera
1992	Bartonella henselae	Cat-scratch disease; bacillary angiomatosis
1994	Sabia virus	Brazilian haemorrhagic fever
1995	Hepatitis G virus	Parenterally transmitted hepatitis
1995	Human herpesvirus-8	Kaposi sarcoma in AIDS patients
1996	TSE causing agent	New variant Creutzfeldt-Jakob disease
1997	Avian influenza (Type A:H5N1)	Influenza
1999	Nipah virus	Inflammation of the brain
2003	SARS-CoV	SARS

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